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NASAL GLANDS

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It is surprising that the numerous papers written on the subject of normal and pathologic nasal mucous membrane dwell so superficially on the subject of nasal glands. In view of this underemphasis the following studies undertake to set forth the importance of the nasal glands under normal and pathologic conditions. These studies are not offered as solving academic problems of histology and pathology. Rather, they are intended to clarify the understanding of certain nasal symptoms.

MATERIAL

The material studied consisted of (1) serial sections through the nose of a newborn infant; (2) serial sections through the upper part of the nose of a patient who succumbed to rhinogenic meningitis, and (3) 300 sections of nasal and paranasal mucous membranes obtained from 100 patients. The majority of the specimens were stained with hematoxylin and eosin; supplementary stainings were by the Mallory-Heidenhain and the Van Gieson method.

ANATOMY

According to Peter,[1] the first anlage of the nasal glands can be found in the tenth week of embryonal life. Further development is apparently very rapid, since in a newborn infant a great number of glands can be found. In the material from a newborn infant which I studied, the large number of glands was indeed surprising. An unusually large number have been found in the tuberculum septi. Kubo [2] showed that there is a considerable accumulation on the mesial surface of the inferior

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1. Peter, K.: Vergleichende Anatomie und Entwicklungsgeschichte der Nase, in Denker, A., and Kahler, O.: Handbuch der Hals-Nasen-Ohrenheilkunde, Berlin, Julius Springer, 1925, vol. 1, pt. 1, p. 95.

2. Kubo, J.: Zur Frage des normalen Zustandes der unteren Nasenmuscheln des Menschen, Arch. f. Laryng. u. Rhin. **19**:191, 1907.

turbinate, while on the inferior and lateral surfaces the glands are poorly developed. The excretory ducts were found to be very short, with many excretory ducts emptying on the surface. Kallius [3] revealed that all glands in newborn infants are albuminous. Kubo [2] was unable to find mucus either in the epithelium or in the glands.

In adults, there are more glands in the nasal mucous membrane than in the mucous membrane of the paranasal sinuses. Sappey [4] estimated that there are one hundred and fifty glands per cubic millimeter.[5] In the tuberculum septi and on the floor of the nose the glands are so piled

Fig. 1.—Wax model of nasal gland (from Maziarski [8]).

up that they produce swellings of the mucous membrane. Occasionally, openings as large as pinpoints (representing the excretory ducts of the glands) can be seen. A great number of glands are found in the inferior

3. Kallius: Sinnesorgane, in von Bardeleben, K.: Handbuch der Anatomie des Menschen, Jena, Gustav Fischer, 1905, vol. 5, p. 12.

4. Sappey, cited by Schumacher: Histologie der Luftwege und der Mundhöhle, in Denker, A., and Kahler, O.: Handbuch der Hals-Nasen-Ohrenheilkunde, Berlin, Julius Springer, 1925, vol. 1, pt. 1.

5. Billroth, cited by Okada.[31]

turbinate. Here, the number of glands decreases progressively toward the epipharynx.[6]

The mucous membrane of the olfactory area is smooth. Conversely, the membranes of the turbinates, of the lateral wall of the nose and of the inferior part of the septum show sulci. These sulci occasionally

Fig. 2.—Nasal mucous membrane from a woman 28 years old who had frequent nosebleeds and who died of Banti's disease. *E* indicates columnar epithelium with many goblet cells; *B*, thickened basement membrane, and *G*, a glandular accumulation with a marked periglandular infiltration, containing resting albuminous and mucous glands. The excretory duct empties on the surface; its cuboid cells extend into the epithelium.

penetrate deeply into the mucous membrane and on cross section present the appearance of crypts. Between the sulci are folds of varying length.

6. Oppikofer, E.: Beiträge zur normalen und pathologischen Anatomie der Nase und ihrer Nebenhöhlen, Arch. f. Laryng. u. Rhin. **19**:28, 1907.

Zuckerkandl [7] stated that the nasal glands empty into the crypts, while Kallius [8] stated that these folds and crypts may increase under pathologic conditions. My findings support the statements of Zuckerkandl and Kallius.

Maziarski [8] constructed a wax model of a mucous gland of the nose (fig. 1), which shows that the chief excretory duct passes into a glandular tube with many branches, the walls and ends of which are covered by bullet-like or egglike alveoli. The excretory duct may show alveolar excrescences. Consequently, the nasal glands are alveolotubular, the diameter of the alveoli being 0.045 to 0.11 mm.

In the sections from the nose of a newborn infant which I studied, the excretory ducts empty into shallow sulci of the mucous membrane.

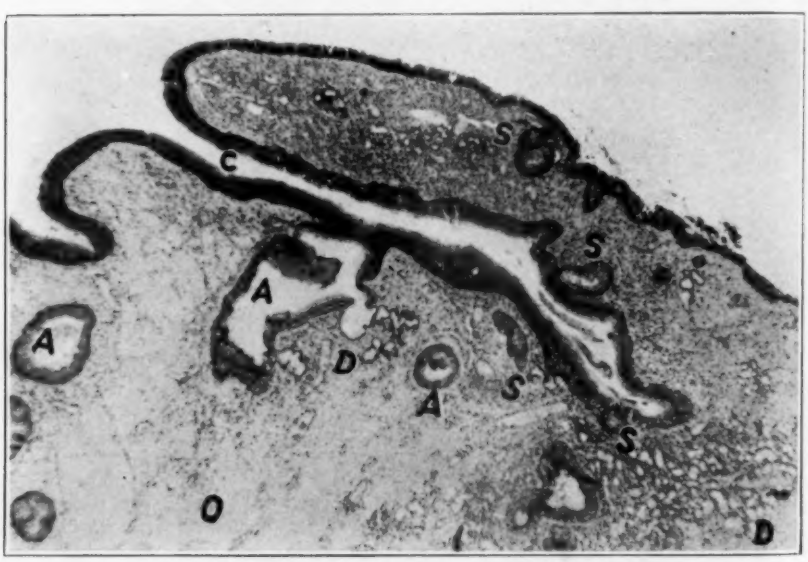

Fig. 3.—Mucous membrane of the right middle turbinate from a man 53 years old with hyperplastic rhinitis and pansinusitis of allergic origin. *C* indicates the crypt; *A*, a dilated excretory duct with columnar epithelium and goblet cells; *S*, active mucous glands; *D*, exhausted glands, and *O*, edematous stroma with many lymphocytes, plasma cells, eosinophils and neutrophils.

As I mentioned in an earlier paragraph, many emptying secretory ducts can be observed in every specimen. In adults one sees a lesser number of excretory ducts and one meets but occasionally with an excretory duct emptying on the surface. Consequently, one must conclude that in post-

7. Zuckerkandl, E.: Normale und pathologische Anatomie der Nasenhöhle und ihrer pneumatischen Anhänge, ed. 2, Vienna, W. Braumüller, 1893, vol. 1.

8. Maziarski, S.: Ueber den Bau und die Einteilung der Drüsen, Anat. Hefte **18**:173, 1902.

embryonic life there is a marked increase in the length of the excretory ducts and an increase of mucous membrane between their openings. Rarely does one find few alveoli (most frequently, albuminous glands), which are situated close to the epithelium and empty into a narrow and short excretory duct, which, in turn, perforates the basement membrane. If the epithelium is of the columnar stratified type, the cuboid cells of the excretory duct can be seen within the epithelium (fig. 2). However,

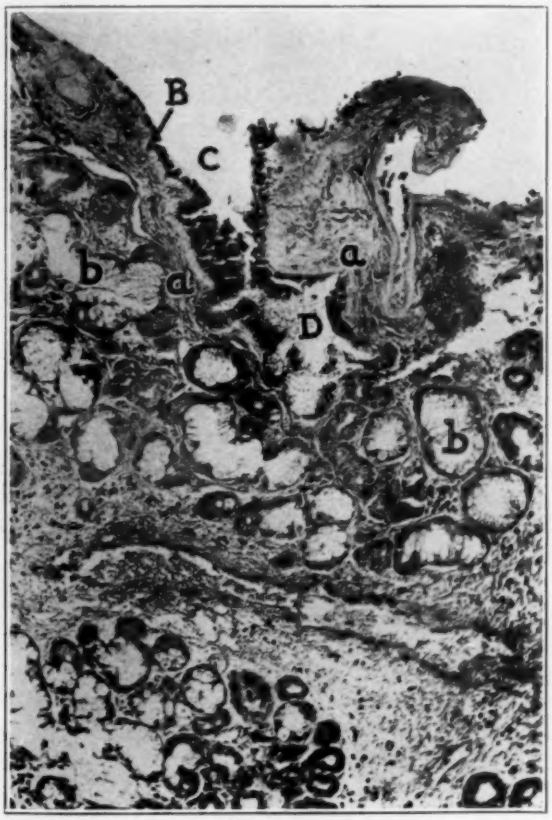

Fig. 4.—Mucous membrane from the middle meatus of a woman with hyperplastic rhinitis. *B* indicates thickened basement membrane, which ends in *a—a*; *C*, the crypt; *D*, an excretory duct with glandular alveoli, and *b*, active mixed type glands with Giannuzzi demilunes.

these findings are exceptions. As a rule, the excretory ducts empty not on the surface but into crypts (figs. 3 and 4).

HISTOPHYSIOLOGY

The nasal mucous membrane contains mucous, albuminous and mixed type glands. In general, it is not possible to say whether the mucous or

the albuminous glands prevail; at any rate, under normal conditions the mixed type glands (viz., the mucous glands with Giannuzzi demilunes) are definitely in the minority. The formation of secretion takes place within the glandular cells in the same way that it does in the salivary glands. The activity of the nasal glands is dependent on stimulation by way of the sympathetic and parasympathetic nervous systems. However, these relations have not been studied sufficiently. Aschenbrandt[9] (1885) found that in rabbits electric irritation of the maxillary nerve increases the activity of the nasal glands and produces a clear watery secretion, while irritation of the sphenopalatine ganglion produces a tenacious opalescent mucus. These facts led to the conclusion that the maxillary nerve contains chiefly parasympathetic fibers, and the sphenopalatine ganglion, sympathetic fibers.

After the secretion is formed, the surface membrane of the muciferous cells ruptures and the secretion is forced into the lumen. The secretion of the albuminous cells is pushed partially into the lumen and partially into the secretory capillaries which lie between the cells and empty into the lumen of the gland. The secretion is transported into a crypt or to the surface of the mucous membrane by the contraction of the myoepithelial basket cells and by the vis a tergo due to the newly formed secretion.

It goes without saying that it is more difficult to transport the tenacious mucus than the watery secretion of the albuminous glands. Consequently, the slightest obstacle to the movement of the secretion leads to retention of the mucous rather than of the serous secretion. Therefore, my attention has been directed chiefly toward the mucous glands, inasmuch as these are easier to examine microscopically than are the albuminous glands. This does not imply that there is much difficulty in recognizing an albuminous gland. However, for a proper understanding of glandular activity it is not sufficient to speak of mucous and albuminous glands per se. Rather, it is necessary to determine as far as possible the stage of activity of these glands. In undertaking such a determination, one must realize that the material on which this work is based is comprised of slides made from routine pathologic specimens that have been passed through the laboratory and therefore is not ideal for microscopic study. Anesthesia and surgical treatment change these very delicate structures, so that artefacts must be expected. Different stages of glandular activity can be recognized:

1. *Albuminous Glands.*—The specimens do not permit an exact distinction between resting and active albuminous glands. In both

9. Aschenbrandt, cited by von Skramlik, E.: Die Physiologie der Luftwege, in Bethe, A.; von Bergmann, G.; Embden, G., and Ellinger, A.: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1925, vol. 2.

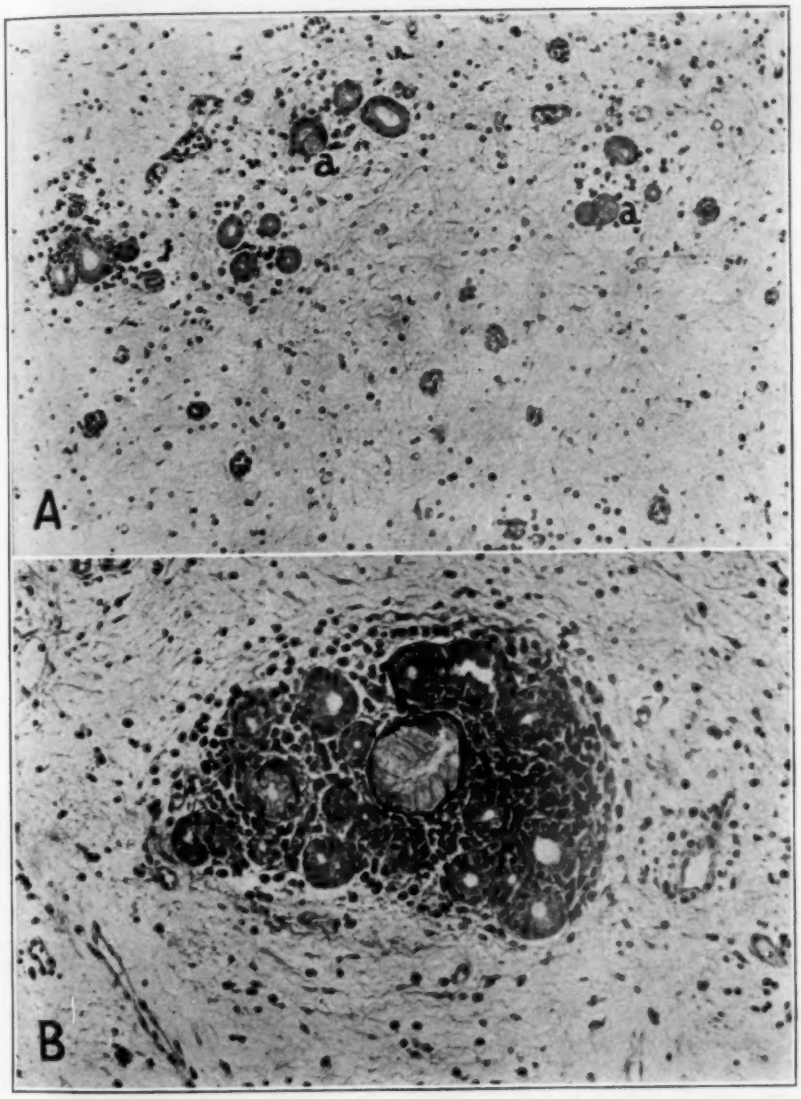

Fig. 5.—Photomicrographs of sections of a nasal polyp from a woman 45 years old who had hyperplastic rhinitis and pansinusitis of allergic origin. In *A*, glandular tubules and excretory ducts are seen scattered throughout the edematous tissue. There are no active mucous glands. At *a* are seen albuminous glands with vacuoles. In *B* an accumulation of glands with a marked periglandular infiltration is seen. In the center one active mucous gland compresses an adjacent albuminous gland. The other tubules are resting albuminous and mucous glands.

stages the gland is small (an active gland is a little larger) and the lumen very narrow. The cells of the gland are small. In the resting stage they take a red and purple stain and in the active stage, a lighter stain. The nucleus is round and close to the base of the cell. The secretory capillaries can be seen only occasionally.

2. *Mucous Glands.*—A resting mucous gland is larger even than an active albuminous gland. The lumen is larger than that in an albuminous gland. The protoplasm has a light, reddish blue tint. The nucleus lies

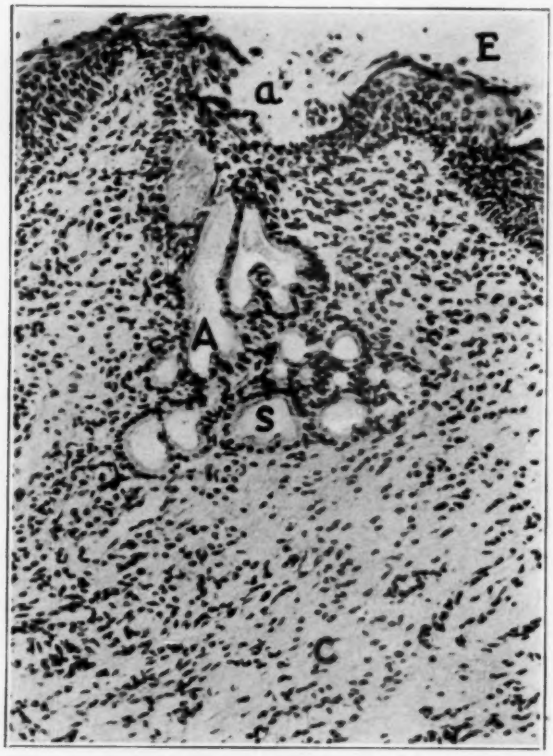

Fig. 6.—Biopsy specimen from the middle turbinate of a women 57 years old. Squamous cell carcinoma of the gingiva had perforated into the maxillary sinus and there was subsequent inflammation of the maxillary sinus. *C* indicates connective tissue infiltrated by lymphocytes, plasma cells and but few eosinophils; *S*, exhausted mucous glands; *A*, three dilated excretory ducts, which contain mucus staining with eosin, and *E*, squamous epithelium, which shows a depression (*a*) at the openings of the excretory ducts.

at the base of the cell and frequently is of an oval shape. Many authorities claim that the cells of resting mucous glands cannot be distinguished from the cells of albuminous glands unless one can visualize the secretory capillaries between the albuminous cells. The distinction

is not easy. Nevertheless, the color of the protoplasm, the form and location of the nucleus and the size of the cell have aided me in making a tentative diagnosis.

In an active mucous gland the nucleus is flattened by the secretion and pushed toward the base of the cell. The entire cell is enlarged and has either a columnar or a rounded form. In a tubule which contains only muciferous cells, either all of the cells or only a part of them are loaded with mucus. However, the boundaries of the individual cells can be distinguished easily. When the cells are loaded with mucus (fig. 5 *B*), the lumen of the gland is very narrow.

An exhausted mucous gland has a large irregular lumen, which is either entirely or partially filled with secretion (fig. 6). The secretion does not always present the chemical reactions of mucus. The walls of the gland are covered by different cells. These are either cuboid or cells with a flattened nucleus, but without mucus or other cellular structures, or are higher cells which still contain some mucus.

3. *Excretory Ducts.*—The excretory ducts most frequently empty into crypts of the mucous membrane, less often on the free surface. One often reads the statement that in the emptying part of the excretory ducts there is ciliated columnar epithelium. This statement is not correct. The walls of the excretory ducts consist of one or two layers of cuboid or higher cells, which may produce mucus under pathologic conditions (fig. 7). If an excretory duct has a large lumen and empties into the bottom of a crypt which is covered by ciliated columnar epithelium, one certainly gets the impression that the ciliated epithelium covers the crypt as well as the excretory duct. However, there is a sharp margin between the cuboid epithelium of the duct and the columnar epithelium of the crypt, even more noticeable when the mucous membrane contains a thickened basement membrane. In this event the thickened basement membrane ends at the boundary between the crypt and the duct (figs. 4 and 8). If the excretory duct has a narrow lumen, the margins between crypt and duct are even more marked, since the duct perforates the basement membrane before emptying into the crypt.

These are the glandular structures which can be found in the mucous membrane of a normal nose. According to Schmincke,[10] the cells of normal nasal glands are capable of expelling their entire secretion, and as they become exhausted, they lose their specificity and appear as indifferent cells without a characteristic nucleus and without a characteristic protoplasm.

I cannot quite agree with Schmincke.[10] It is, of course, difficult to state what a normal condition of the mucous membrane of the nose is.

10. Schmincke, A.: Zur Kenntnis der Drüsen der menschlichen Regio respiratoria, Arch. f. mikr. Anat. **61**:233, 1903.

This has been pointed out recently by Fabricant.[11] The question is even more difficult to answer when one considers that Aschoff (quoted by Runge [12]) occasionally found signs of irritation of the nasal mucous membrane in newborn infants, while Polis [13] frequently found such signs after the seventh day of extrauterine life. However, this question concerns not only the nasal mucous membrane but also the mucous membrane of the entire respiratory and the entire digestive tract and the skin. In other words, it concerns all membranes which are influenced by chronic irritation.

It is not difficult to recognize an acute inflammation of these membranes. It is, however, extremely difficult to distinguish between

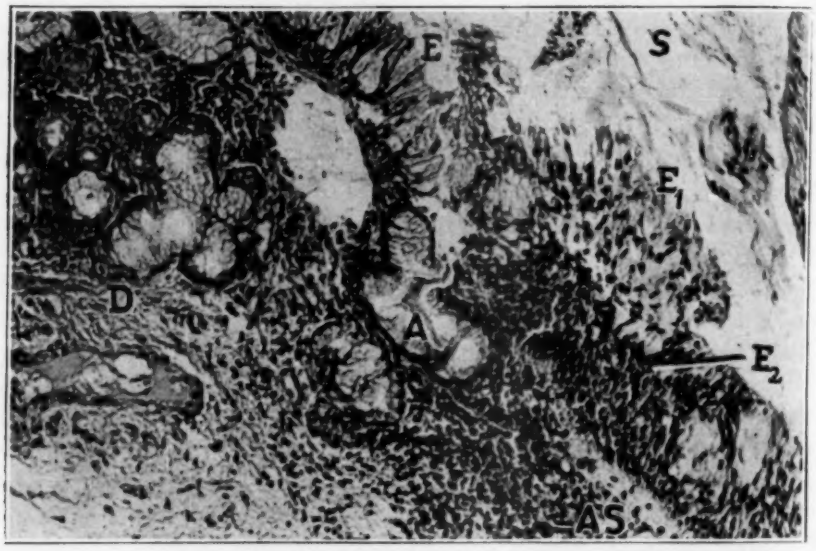

Fig. 7.—Biopsy from the middle turbinate of a man 58 years old with carcinoma of the piriform sinus. *E* indicates columnar epithelium with many goblet cells; E_1, desquamating columnar epithelium; E_2, regenerated epithelium without goblet cells; *S*, expelled mucus and desquamated epithelium; *A*, an excretory duct covered with goblet cells only (note the mucous plug in the opening of the duct); *AS*, adenoid tissue below the epithelium, and *D*, albuminous and mixed type glands, the latter with Giannuzzi demilunes within the adenoid layer.

chronic irritation, a physiologic state, and chronic inflammation, a pathologic state. The difficulty becomes obvious when one considers

11. Fabricant, N. D.: The Nasal Biopsy, Laryngoscope **50**:527, 1940.

12. Runge, H. G.: Die entzündlichen Erkrankungen der Nase und ihrer Nebenhöhlen, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1928, vol. 3, p. 67.

13. Polis, cited by Kubo.[2]

periglandular infiltration, found in the vicinity of glands situated in the deeper layer of the mucous membrane as well as in that of glands situated in the subepithelial, adenoid layer. Occasionally, one even sees a lymphocyte (never a polymorphonuclear or an eosinophil leukocyte) penetrating the basal membrane of an albuminous gland and appearing between the glandular cells. Periglandular infiltration is usually con-

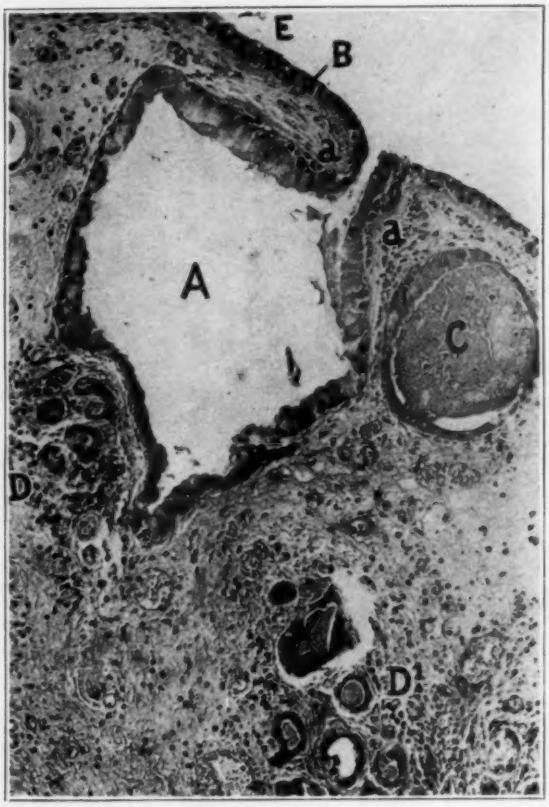

Fig. 8.—Section of a nasal polyp from a man 34 years old with polyposis nasi. *A*, indicates a markedly dilated excretory duct with many goblet cells; *E*, squamous epithelium; *B*, thickened basement membrane, which ends in *a*—*a*; *C*, cyst with cuboid cells producing mucus; *D*, albuminous glands, and D_1, albuminous glands with secretion.

sidered to be a sign of chronic inflammation. Schall [14] pointed out that he was much impressed by the marked periglandular infiltration in chronic inflammation. However, there are some doubts as to whether

14. Schall, L. A.: The Histology and Chronic Inflammation of the Nasal Mucous Membrane, Tr. Am. Acad. Ophth. **37**:382, 1932.

periglandular infiltration indicates chronic inflammation. Zimmermann [15] reported finding an accumulation of plasma cells and lymphocytes in all human salivary glands, a finding which is particularly marked in embryos and in newborn infants. It is true that the accumulation of lymphocytes, as a rule, surrounds the excretory ducts rather than the glands. Nevertheless, this finding proves that periglandular infiltration is not always due to chronic inflammation. In fact, Maximow [16] proved with sound arguments that the plasma cells probably carry some nutritive material from the blood to the glandular cells which the latter require for the production of secretion.

Considering these facts, one cannot call periglandular infiltration a certain sign of chronic inflammation. I believe that it plays a role in the metabolism of the glands, unless there is an extremely intense infiltration or many neutrophils or eosinophils between the lymphocytes or an invasion of a gland by lymphocytes (fig. 9). At any rate, in adults the complete absence of lymphocytic infiltration of the nasal glands (figs. 5 *A*, 6, 10 and 11) is more indicative of a pathologic state, particularly of atrophy of glands, than is its presence.

Under normal circumstances only resting and active glands and occasionally an exhausted gland have been found in the nasal mucous membrane. This suggests that under normal conditions the nasal glands as a rule do not expel their entire secretion so that they soon become exhausted. The glands are almost continually active, unless they are poisoned by pilocarpine. There is only a low grade activity of the nasal glands, producing an inconspicuous secretion. In this respect the nasal glands do not differ from the salivary glands. The contrary finding of Schmincke [10] can be explained by the fact that he examined not a normal membrane but one with "catarrhal" changes.

In closing this portion of the discussion, one finding which probably lies on the borderline between physiology and pathology should be mentioned. In 6 cases onkocytes (Hamperl [17]) or pyknocytes (Zimmermann [15]) were found. Brunner and Wall [18] found these cells in a case of carcinomatosis of the nasal mucous membrane. The cells are very large. The protoplasm shows fine granules, more resistant than the granules of normal glandular cells, and stains with eosin as intensely as eosinophilic leukocytes (fig. 12). The nucleus is relatively small,

15. Zimmermann: Die Speicheldrüsen der Mundhöhle und die Bauchspeicheldrüse, in von Möllendorff, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1927, vol. 5, pt. 1, p. 61.

16. Maximow, A.: Beiträge zur Histologie und Physiologie der Speicheldrüsen, Arch. f. mikr. Anat. **58**:1, 1901.

17. Hamperl, H.: Ueber das Vorkommen von Onkocyten in verschiedenen Organen und ihren Geschwülsten, Virchows Arch. f. path. Anat. **298**:326, 1937.

18. Brunner, H., and Wall, J. W.: Carcinomatosis of the Nasal Mucous Membrane, Ann. Otol., Rhin. & Laryng. **49**:688, 1940.

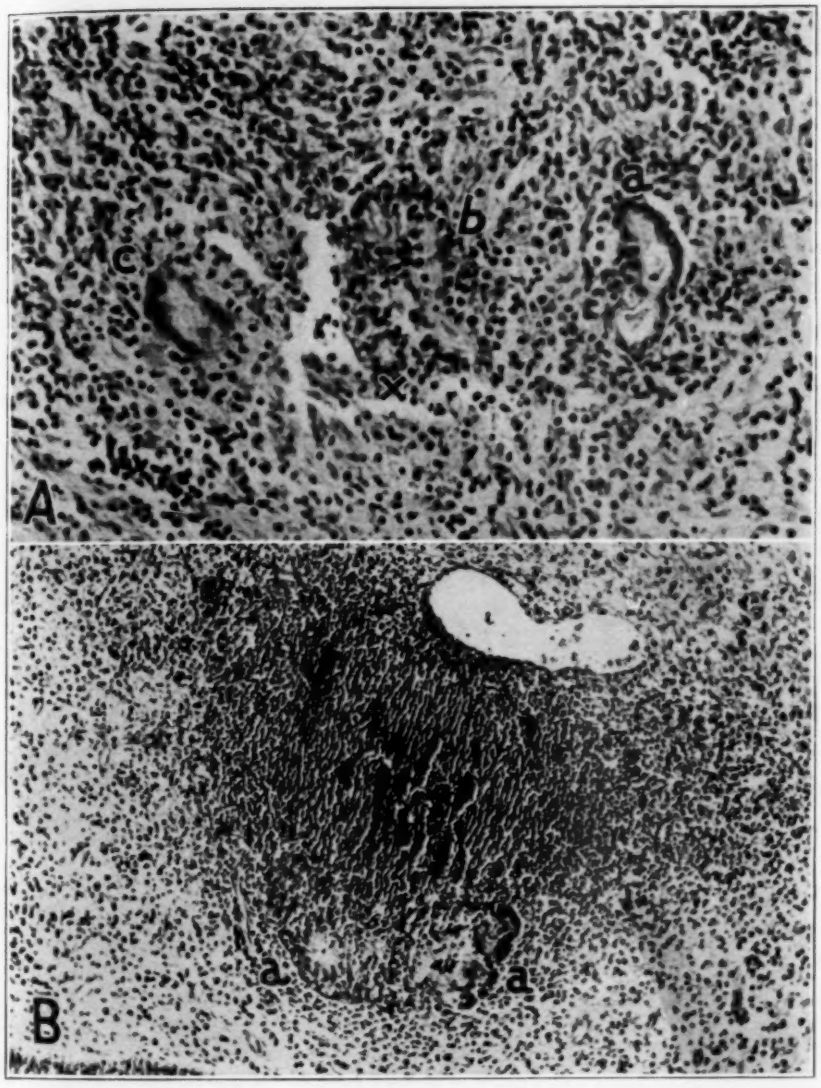

Fig. 9.—*A*, section of a gumma septi from a woman 43 years old. At *a* are seen albuminous glands containing some secretion. On one side lymphocytes and plasma cells invade the gland, while on the other the cells of the gland are flattened and there are chromolytic nuclei. At *b* is seen an accumulation of glands entirely destroyed by lymphocytes and plasma cells. At *x* a glandular tubule can be recognized. At *c* the glands have been changed into what looks like a giant cell. Note the lumen and the secretion within that formation. *B*, mucous membrane from a maxillary sinus, which is infiltrated by lymphocytes and plasma cells, from a woman 38 years old with purulent inflammation of the maxillary sinus. In the region designated by *a*—*a* the glands have been destroyed by lymphocytes and plasma cells.

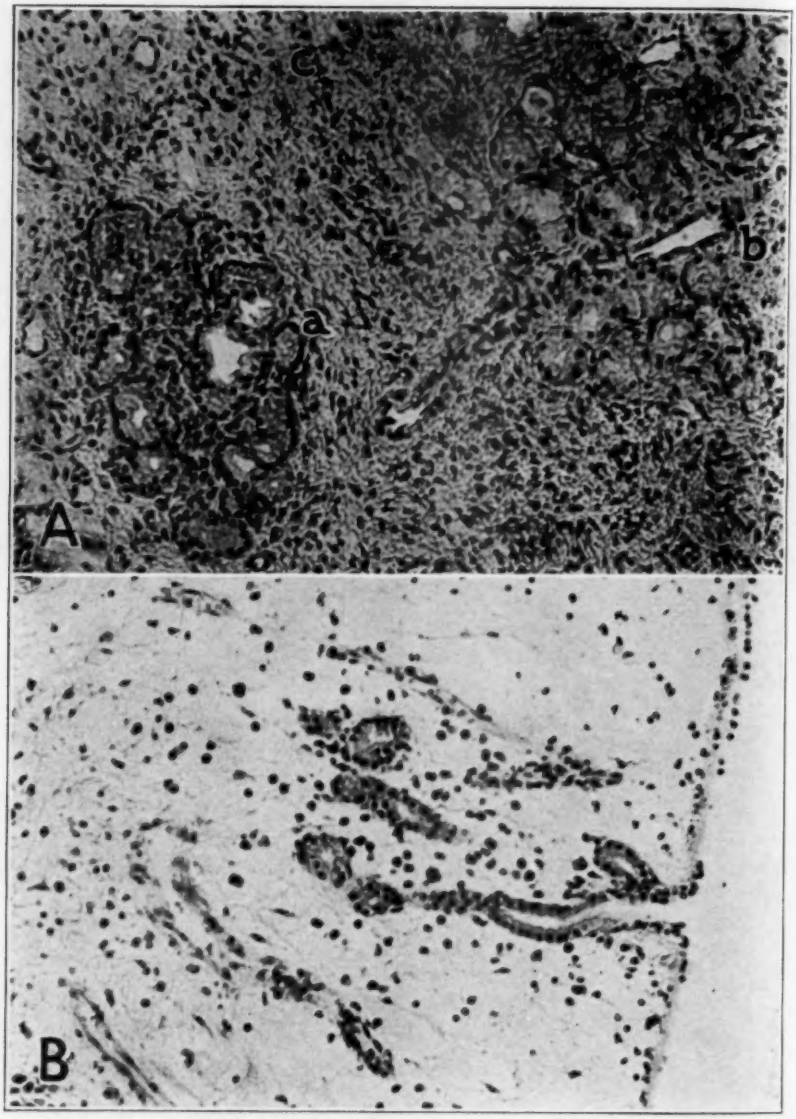

Fig. 10.—*A*, biopsy specimen from an ethmoid sinus of a woman 45 years old with a history of syphilitic infection who had hyperplastic ethmoiditis. At *a* is seen an accumulation of normal resting glands with a definite periglandular infiltration; at *b*, an accumulation of degenerated glands without periglandular infiltration, and at *c*, loose connective tissue with lymphocytes and plasma cells. *B*, excretory duct emptying at the surface of a polyp (from the case illustrated by figure 5). Few albuminous glands which do not form a typical accumulation and which are atrophic can be seen. The boundaries of the glands, as well as those of the individual glandular cells, are not distinct, and there is an irregular arrangement of nuclei. There is no definite periglandular infiltration, but there is a diffuse infiltration of the tissue by lymphocytes and plasma cells and particularly by eosinophils.

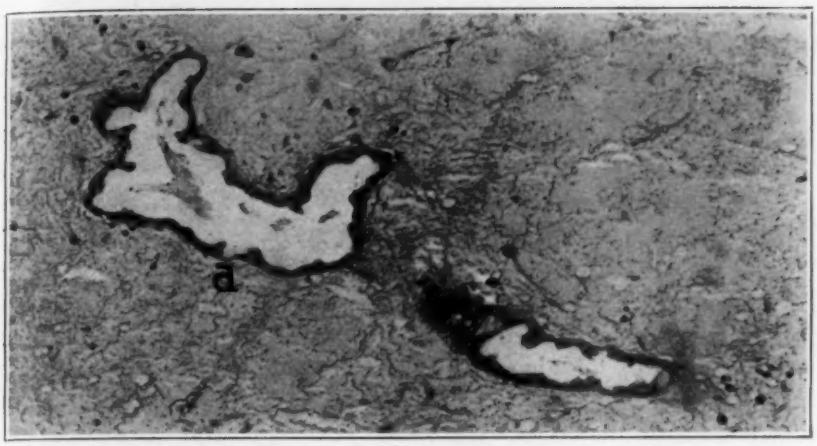

Fig. 11.—Atrophic glands within a nasal polyp from a man 75 years old with polyposis nasi. The glandular cells are cuboid or flattened. The arrangement of the cells is irregular. The nuclei are pyknotic. At *a* the cells are missing.

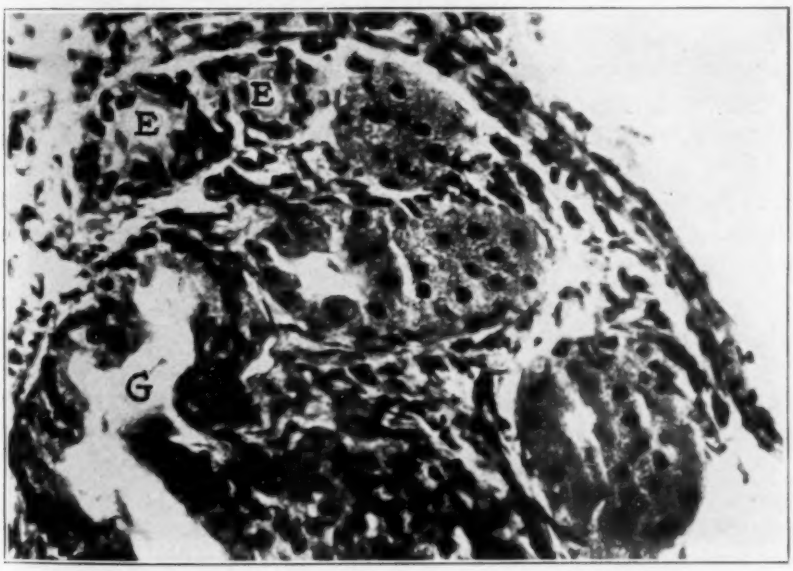

Fig. 12.—Biopsy specimen from the nasal mucous membrane of a man 79 years old with hyperplastic rhinitis and sinusitis. The blood contained 6 per cent eosinophils. *E* indicates excretory ducts and *G*, an exhausted gland. There are four glands consisting only of onkocytes. Three glands are presented in tangential section and one in cross section.

has an irregular shape, is occasionally pyknotic and lies in the center of the cell. These cells can be found between normal cells of the glands or the excretory ducts; they can also surround the entire lumen of a gland or an excretory duct.

The function of these cells is not known. It is noteworthy that I found them most frequently in old persons, viz., in patients between 57 and 79 years of age. The only exception concerned a woman 28 years old, a person who suffered from frequent nosebleed and died of Banti's disease.

PATHOLOGIC CHANGES

Apart from neoplastic diseases, the nasal mucous membrane can be influenced by a great number of pathogenetic factors. Two of these factors are: (1) infection and (2) allergy. It would be erroneous to suppose that these are the only ones. On the contrary, there can be no doubt that such factors as disturbances of the endocrine glands, psychic conditions and different functional stages of the sympathetic and parasympathetic systems exert definite effects on the nasal mucous membrane. General marasmus is important in this respect, since it frequently produces a marked stimulation of the mucous glands (figs. 6 and 7). Since these influences have not all been studied thoroughly, this paper deals only with infection and allergy. Atrophic rhinitis is not included, since the nasal glands as affected by this disease have been studied on numerous occasions by other investigators.

Toxic Atrophy of the Nasal Glands.—Eight cases are included in this category. In 6 of these there was a purulent inflammation of one or more paranasal sinuses. In 5 a specimen for biopsy was taken from the middle turbinate and in 1 from the mucous membrane of the maxillary sinus. In 1 case there was hyperplastic rhinitis in a syphilitic person and in 1 case a gumma of the septum.

In all of these cases the connective tissue of the mucous membrane was changed to granulation tissue infiltrated by neutrophils, lymphocytes and plasma cells. In that of hyperplastic rhinitis combined with syphilis there were few eosinophils, while in that in which there was a gumma of the septum the neutrophils were missing, although there were giant cells and spotlike areas of necrosis. When the nasal glands and the epithelium are exposed to the toxins originating in the granulation tissue, the first thing that happens is a cessation of mucus production. In such a case the epithelium shows but few goblet cells, while active mucous glands are almost entirely absent. There may be some resting glands or atrophic glands; however, the majority of the glands show definite pathologic changes. The nucleus of a glandular cell loses its chromatin and presents a pale-staining reaction. Seldom does it become pyknotic, for it remains hypochromatic even when a great deal of the gland is

destroyed. The outlines of the protoplasm become indistinct. Further changes can occur in one of two directions:

It is possible for the glandular cells to break down and form protoplasmic disks with indistinct outlines and a garland-like arrangement of the nuclei (fig. 9 *A*). Although this gives the impression of Langhans' giant cell, it is not a giant cell, for it is easy to follow the development of the formations; high magnification shows a lumen within the formation and occasionally some secretion within the lumen.

More frequently, a great number of lymphocytes, plasma cells and occasionally neutrophils invade a dying gland and destroy it entirely (fig. 9 *B*).

A woman 45 years old suffered from impairment of breathing. She gave a history of syphilitic infection. The Wassermann reaction of the blood was negative, although there were neurologic findings suggestive of syphilis of the brain. Examination revealed what was supposed to be hyperplastic ethmoiditis, and a specimen for biopsy was taken from the ethmoid sinus.

Microscopic examination revealed a columnar epithelium with few goblet cells and few migrating leukocytes. There was a definite formation of papillae. The basement membrane was thickened, and the stroma consisted of a loose connective tissue, infiltrated by lymphocytes, plasma cells and a few eosinophils. There were many accumulations of normal glands, with marked periglandular infiltration; the glands were resting, active, exhausted and atrophic mucous glands and, to a great extent, normal albuminous glands. There were many onkocytes. Most striking was the following finding: In some areas the protoplasm of the glandular cells lost its oxyphilia. The cells presented a pinkish tint, their outlines had disappeared and there was only one band of protoplasm, with irregularly distributed nuclei. Since there was not definite periglandular infiltration, it was difficult to recognize these glands within the infiltrated connective tissue (fig. 10 *A*). This finding is not an artefact, inasmuch as close to these degenerating glands normal glands were found. This type of glandular degeneration was observed in only a single case. The causation is not clear.

Atrophy of the Nasal Glands by Glandular Dysfunction.—There is little doubt that allergy is an important factor in producing pathologic changes of the nasal mucous membrane, the so-called "hyperplastic rhinitis." However, there is much doubt as to how often and to what extent allergy is of importance. Hansel [19] emphasized the importance of the presence of eosinophils in nasal secretion for the diagnosis of nasal allergy during exacerbation as well as during quiescence. Furthermore, the accumulation of eosinophils in the tissues of allergic persons has been observed by many workers, while Walsh and Lindsay [20] found such accumulations in nasal polyps.

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19. Hansel, F. K.: Allergy of the Upper and Lower Respiratory Tracts in Children, Ann. Otol., Rhin. & Laryng. **49**:579, 1940.

20. Walsh, T. E., and Lindsay, J. R.: Cytology of Nasal Polypi, Arch. Otolaryng. **20**:649 (Nov.) 1934.

Among 68 of the cases in which my material was obtained and in which the presence of allergy was suspected because of the history and clinical findings, there was definite eosinophilia in only 14. In 22 there were large numbers of eosinophils, but since there were many lymphocytes and plasma cells, a diagnosis of eosinophilia per se was not made. Perhaps other investigators would speak of eosinophilia in some of these cases, but this is dependent on the subjective judgment of the examiner. Hansel [19] stated the belief that in an evaluation of the cytologic findings, individual interpretation must be correlated with all of the clinical facts.

Because of such uncertainty the concepts of "minor allergy" (Vaughan [21]), "equivocal allergy" (Semenov [22]) and "borderline allergy" (Fox, Harned and Peluse [23]) have been advanced. These terms arouse suspicion of an "allergia sine allergia." Notwithstanding such confusion, allergy plays an important role in nasal disease, and definite eosinophilia certainly points in this direction. Inasmuch as it is rather difficult to draw a sharp line of distinction between what one knows and what one believes one knows, and since this paper does not deal with etiology, but with the pathologic changes of the nasal glands, this subject is discussed so far as (*a*) the hyperplastic mucous membrane of the nose and the paranasal sinuses and (*b*) the nasal polyps are concerned, regardless of whether the allergy is of primary or secondary importance.

Joseph [24] found that in nasal polyps the glands were located either at the periphery or in the center of the polyp. The glands were normal, hypertrophic, atrophic or absent. Goodale [25] found a proliferation of glands in hypertrophic rhinitis. In vasomotor rhinitis, the glands evidenced a moderate increase in number and size, but there was apparently no increase in the amount of mucous degeneration and there were no signs of increased activity in the albuminous glands. In polyps, few glands were found. In "catarrhalic" inflammation of the nasal mucous membrane, Hajek [26] observed an increased production of mucus in the glands as well as in the secretory ducts. Tobey [27] spoke of a pale and a red mucous membrane in vasomotor rhinitis. In the pale mucous

21. Vaughan, W. T.: Minor Allergy, J. Allergy **5**:184, 1934.

22. Semenov, H.: The Surgical Pathology of Nasal Sinusitis, J. A. M. A. **111**:2189 (Dec. 10) 1938.

23. Fox, N.; Harned, J. W., and Peluse, S.: Borderline Allergy, Arch. Otolaryng. **31**:502 (March) 1940.

24. Joseph, cited by Suchannek, H.: Pathologie der Atmungsorgane, Ergebn. d. allg. Path. u. path. Anat. **10**:380, 1906.

25. Goodale, J. L.: A Contribution to the Study of the Secreting Mechanism of the Nose, Tr. Am. Laryng. A. **26**:27, 1904.

26. Hajek, M.: Sogenannte intraepitheliale Drüsen der Nasenschleimhaut, Arch. f. Laryng. u. Rhin. **17**:95, 1905.

27. Tobey, cited by Weille.[39]

membrane are active mucous glands and dilated serous acini, while red mucous membrane contains serous but not dilated glands. Mullin and Ball,[28] as well as Schall,[14] found dilated glands in the edematous type of chronic inflammation. Weille [29] observed a pinkish-staining secretion in some dilated glands and excretory ducts. This may cause obstruction, with subsequent development of a cyst. In allergy, Hansel [30] found dilatation, compression and atrophy of glands. Obstructed gland acini and ducts may appear as large cysts filled with secretion. In areas where the tissue is compact, the glands appear compressed or atrophied, the connective tissue is firm and there is less infiltration with eosinophils. An alteration of glands and other tissues of the tunica propria occurs as a result of the stretching of the membrane caused by the weight of polypoid formation. Localized masses of glands may appear stretched or elongated. Stretching of the deep layers places the connective tissue under tension and causes compression of glands and blood vessels.

One cannot expect absolute uniformity of findings as far as the glands are concerned. It must be borne in mind, first, that only a small part of the mucous membrane is at the pathologist's disposal for microscopic examination; second, that the microscopic findings are dependent on the stage of glandular activity; third, that the microscopic findings are dependent on whether the biopsy is made in a quiescent stage or during an exacerbation of the disease and, fourth, that the findings are dependent on whether the patient suffered from a great or a small number of exacerbations. Consequently, a certain degree of variability of findings must be expected in the examination of hyperplastic mucous membrane of the nose as well as in that of polyps.

(*a*) Hyperplastic Mucous Membrane: Biopsy specimens of 40 patients were studied. In only a single instance glands were not found. In 16 instances normal glands were observed; i. e., there were mucous and albuminous glands either in the active or in the resting stage. Exhausted glands were found occasionally, but they were never conspicuous.

In 6 instances there was definite hyperactivity of the glands, particularly of the mucous glands. Occasionally, the glands were dilated to such an extent that the wall of a crypt was bulged into the lumen when such a gland was adjacent to the crypt (fig. 3). The entire gland consisted of muciferous cells, the nuclei being flattened to a

28. Mullin, W. V., and Ball, R. P.: Studies of Pathologic Tissue Removed from Chronically Infected Nasal Accessory Sinuses, Ann. Otol., Rhin. & Laryng. **37**:182, 1928.

29. Weille, F. L.: Asthma: Pathology of Allergic Tissue as Seen in Nose and Accessory Sinuses, Arch. Otolaryng. **12**:785 (Dec.) 1930.

30. Hansel, F. K.: Clinical and Histopathologic Studies of the Nose and Sinuses in Allergy, J. Allergy **1**:43, 1930.

maximal degree. The lumen of the gland was absent; however, the outlines of the cells could be recognized. Some of the specimens were entirely filled with such hyperactive glands; in other specimens many Giannuzzi demilunes were seen. Frequently, the cells of the excretory ducts exhibited marked production of mucus. In the presence of enormous production of mucus the albuminous glands were of little importance. In some cases the epithelium presented marked production of mucus, while in other cases it did not. When the hyperactive glands expelled their secretion, they fell back not into the resting stage but into the exhausted stage. In fact, exhausted glands were found in many of the specimens studied (fig. 13).

Fig. 13.—Nasal mucous membrane from a woman 58 years old with hyperplastic rhinitis. *E* indicates squamous epithelium; *D*, exhausted mucous and mixed type glands, and *L*, Giannuzzi demilunes.

When the hyperactive glands could not expel their secretion, the findings were quite different. Thus, the hyperactivity became combined with retention of secretion. Retention can occur very easily, particularly when a gland empties into a crypt and the latter, in turn, becomes obliterated by the surrounding edema or obstructed by desquamated epithelium (fig. 4), debris and mucus. Under these circumstances two things can occur: The dilatation of the gland increases and simultaneously the outlines of the muciferous cells disappear. In the cross section one then sees that the basilar membrane of the gland contains a mixture of mucus, nuclei and protoplasm (fig. 14 *A*). It is interesting

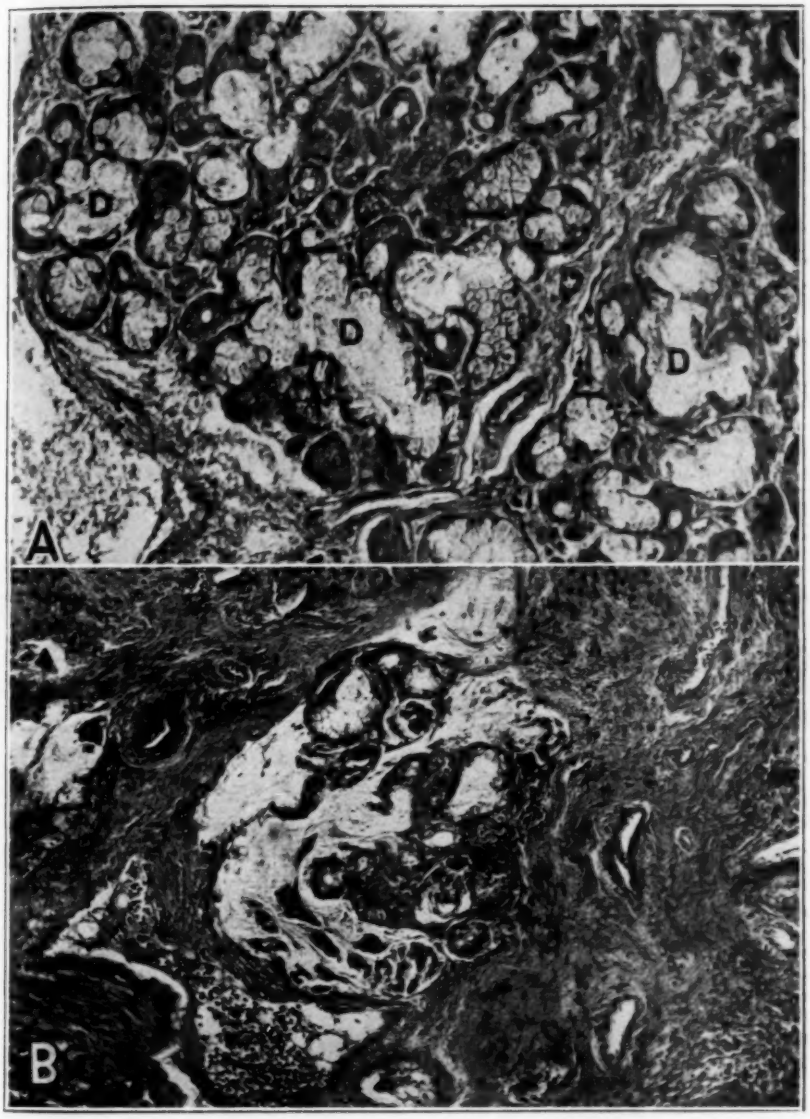

Fig. 14.—*A*, mucous membrane from the middle meatus of a woman 30 years old with hyperplastic rhinitis. *D* indicates dilated mucous and mixed type glands close to rupture. *B*, nasal mucous membrane from a woman 31 years old with hyperplastic rhinitis and pansinusitis on the left side. Within the connective tissue there are ruptured mucous glands. Note the remnants of glands and the relatively well preserved albuminous glands within the mucus.

to note that this finding is the same as that which Maximow [16] described in the retrolingual glands of dogs when he dissected the secretory nerve. However, this is not the final stage of the hyperactivity of nasal glands. If the retention continues, the glandular vesicle ruptures and the mucus, with the cellular debris, pours out into the surrounding connective tissue (fig. 14 *B*). Many years ago Okada [31] found mucus within the connective tissue. However, he found it only in the neighborhood of cysts and did not observe that it was coming from hyperactive glands.

The second condition which arises from hyperactivity and retention in mucous glands is the formation of secreting cysts. McGregor,[32] who contributed an excellent article concerning these and other cysts of the maxillary sinus, expressed the belief that the prevalent concept that secreting cysts are mechanically produced by obstruction alone is not an adequate explanation and that damage to the cilia of the gland tubules due to infection is the primary cause of these cysts. In this respect I cannot quite agree with McGregor, for the following reasons: First, there are no cilia either in the glandular tubules or in the excretory ducts, for there are cilia only in the crypts where the excretory ducts drain. Second, I observed cysts only in cases apparently without infection. Third, Mosher [33] and Schall [14] found that in practically every chronically infected antrum, no matter how foul the secretion, the mucous membrane shows active cilia. If one does not consider the retention of mucus a conditio sine qua non (as Billroth [5] did not), at any rate, the development of cysts is seen to be due mainly to retention which allows the secretion to pile up from behind, causing increased pressure in the crypts and in the excretory ducts. Thus, the marked enlargement of the excretory duct in figure 8 can be explained readily. Since the epithelium of the crypts and the excretory ducts is by far more resistant than the highly organized epithelium of the glandular tubules, it can endure the internal pressure without becoming entirely flattened. Thus, the mucous membrane frequently contains cysts, the walls of which are covered by a cuboid or even a higher epithelium, occasionally producing mucus (fig. 8). All of these cysts probably are derived from the crypts or from the excretory ducts. In the first instance they frequently are surrounded by a basement membrane.

As I have already mentioned, the glandular tubules rupture when the pressure from within increases, and this phenomenon occurs only

31. Okada, W.: Beiträge zur Pathologie der sogenannten Schleimpolypen der Nase, nebst einigen Bemerkungen über Schleimfärbungen, Arch. f. Laryng. u. Rhin. **7**:204, 1898.

32. McGregor, G. W.: The Formation and Histologic Structure of Cysts of the Maxillary Sinus, Arch. Otolaryng. **8**:505 (Nov.) 1928.

33. Mosher, H. P.: Nasal Mucous Membrane, Tr. Am. Acad. Ophth. **37**:440, 1932.

occasionally to excretory ducts. It takes place if the pressure increases rapidly and if the glandular tubules are embedded in a more or less firm connective tissue. As pointed out by McGregor,[32] ease of distention of the surrounding structures is an important factor in the formation of cysts. Consequently, only a small amount of pressure is necessary to expand a glandular tubule without rupturing it, provided the tubule lies not in a bed of firm connective tissue but in a loose edematous mucous membrane, as for example, in a nasal polyp. Such cysts have greatly flattened epithelium, as will be described later in this discussion.

Hyperactivity of glands and retention of secretion lead to (1) exhaustion and (2) destruction of the glands and (3) to formation of cysts. These pernicious effects of hyperactivity and retention relate practically only to the mucous glands, not to the albuminous glands of the nose. This is in exact accordance with the findings of Maximow [16] concerning the "paralytic secretion" of the salivary glands. That these effects are pernicious is obvious so far as the destruction of glands and the formation of cysts are concerned. However, exhaustion is an equally pernicious effect. There is but a difference of degree between exhaustion and atrophy of glands, inasmuch as an exhausted gland can recover, while an atrophic gland is not capable of doing so. In both of these circumstances the gland is definitely dilated and the lumen of the gland is irregular. The flattening of the cells in an atrophic gland is more intense and extensive and the periglandular infiltration is, as a rule, absent. (Compare figures 11 and 13.) In consideration of these facts the finding of atrophic glands in hyperplastic mucous membrane is to be expected. As a matter of fact, in 17 of my cases resting, exhausted and atrophic glands were the predominant finding.

In summarizing, it is apparent that hyperplastic mucous membrane presents either a normal condition or hyperactivity of the glands with or without retention of secretion, or exhaustion and atrophy of the glands. Because of the great variability there is no reference to the number of eosinophils. In all three conditions there were occasionally many eosinophils and sometimes only a few, although definite eosinophilia was observed most frequently in cases in which there were exhausted and atrophic glands.

(*b*) Nasal Polyps: Forty-five polyps were examined. The main finding characteristic for the polyps is edema. One would assume that this edema drains toward the surface, along the channels of the basement membrane. However, this does not occur; on the contrary, the edema within the polyps stays under high pressure, so that in 1 case the epithelium was ruptured, while in another instance the edema penetrated into the epithelium. The channels of the basement membrane, with the supposition that they are actually present, do not fulfil their task in

polyps, although in but a single case the basement membrane was observed to be exceedingly thick. In 22 cases the basement membrane was of normal thickness; i. e., it could scarcely be visualized, while in the other cases the basement was of normal thickness except in the crypts, where it was, as a rule, slightly thickened.

The first effect exerted on the glands by edema is the complete or almost complete prevention of the production of mucus in the epithelium as well as in the glands. In 16 of the polyps the epithelium, being of squamous, columnar or transitional type, did not present any goblet cells. In the other cases there were some goblet cells, most frequently in the crypts. In only a single case was there such an accumulation of goblet cells as is seen occasionally in hyperplastic mucous membrane.

The same finding holds true for the glands. Occasionally there are active mucous glands. However, in but 1 case a large number of hyperactive, or active, mucous glands was observed. Further characteristics of polyps are (1) sparseness of glands, (2) cyst development and (3) atrophy of glands.

In order to understand these characteristics one must answer the following question: What occurs when edema exerts pressure on the glands? The glandular heaps, as well as the periglandular infiltration, are shredded, and the individual alveoli, together with the lymphocytes and plasma cells, are scattered throughout the edematous area. In figure 5, *A* illustrates a shredded glandular accumulation, while *B* shows within the same polyp a glandular accumulation which has escaped the destructive effect of the edema. Owing to the mechanical effect of the edema, glandular accumulations as well as individual alveoli may even be absent in polyps. In fact, in 17 of the 45 polyps examined there were no glands at all, although large-sized polyps were examined. The importance of this finding becomes obvious when one recalls that of 40 specimens for biopsy taken from hyperplastic mucous membranes there was only 1 without glands, although these specimens contained less tissue than the polyps.

When the glandular accumulations are torn by the edema, the individual alveoli lose their connection with the excretory ducts and must undergo degeneration. This is particularly true as far as the very vulnerable mucous glands are concerned and true to a lesser degree so far as the more resistant albuminous glands are concerned. The latter occasionally present large vacuoles within the protoplasm (fig. 5 *A*). The contents of these vacuoles are not known, because they do not stain with mucicarmine. At any rate, the degeneration finally leads to atrophy (figs. 10 *B* and 11), which, in turn, may lead to complete destruction of the gland or formation of cysts. In the latter event, the walls of the cysts are covered by an extremely flattened epithelium, and the contents consist of an edematous fluid, mucus and cellular

debris. It goes without saying that the cysts which originate in the alveoli as well as those originating in the excretory ducts and epithelial crypts may become infected secondarily.

All of these changes which lead to atrophy of glands and formation of cysts take place more rapidly when the formation of polyps concerns a hyperplastic mucous membrane already containing atrophic glands due to retention and hyperactivity.

COMMENT

Purulent as well as hyperplastic inflammation of the nasal mucous membrane leads to atrophy and destruction of the nasal glands. Consequently, the term "hyperplastic rhinitis" is a misnomer as far as the glands are concerned. There are, rather, hypertrophy and subsequent atrophy of the glands, but not hyperplasia of the glands.

Although the final results of purulent and hyperplastic inflammation are similar, there are some definite differences between the two conditions. In purulent inflammation the glands become atrophic because of toxins and they are invaded by lymphocytes and leukocytes, which destroy them. The destruction goes on rapidly. Mucous glands, as well as mixed and albuminous glands, are affected, although the mucous glands are first involved. There is no formation of cysts. A restitution of the glands does not take place.

In hyperplastic inflammation, hyperactivity, retention and edema are the destructive factors. The mucous glands are affected earlier than the albuminous glands. They become dilated, and the cells produce a large amount of mucus. The mucus may be expelled and the glands become exhausted. In this stage restitution is possible. If the glands cannot expel their secretion, the dilation increases, the outlines of the cells disappear and the gland ruptures or forms a cyst originating in an alveolus, in the excretory duct or in one of the crypts into which the excretory ducts drain. The destruction takes place slowly.

The cause of purulent inflammation is clear; it is an infection. What causes hyperplastic inflammation is still a moot question. At any rate, the cause is not uniform. Hyperplastic rhinitis due to tumor of the paranasal sinuses or the epipharynx is one clinical entity. Another, and probably the most common clinical entity, is the hyperplastic rhinitis due to allergy. Consequently, the changes involving the glands in hyperplastic rhinitis must be related to allergy. This is not just a groundless hypothesis, inasmuch as exactly the same changes are found in the glands of the bronchial mucous membrane in cases of allergic asthma. Steinberg and Figley [34] stated that in allergic asthma

34. Steinberg, B., and Figley, K. D.: Pathology of Asthma, J. Lab. & Clin. Med. **13**:921, 1928.

the nuclei of the glandular cells are absent and the outlines of the cells obliterated and that there is hypersecretion of mucus. On the contrary, in bacterial asthma the glands do not show any change except moderate infiltration by mononuclear and polymorphonuclear cells. In the case reported by Kountz and Alexander [35] the mucous membrane of the bronchi presented an epithelium with many goblet cells and the basement membrane was thickened. In some areas the glands were normal; in others they were dilated. Many of their cells had undergone mucoid degeneration, and the alveoli were filled with mucus. The similarity of these findings to those in the nasal mucous membrane of patients with hyperplastic rhinitis is striking. In fact, Kountz and Alexander examined the nasal mucous membrane in one of their patients and found a markedly hyperplastic epithelium, edema and a subepithelial accumulation of lymphocytes and eosinophils. The nasal glands were prominent and undergoing mucoid degeneration.

If one supposes that the concept of allergic asthma holds true, there can be no doubt that the hyperactivity of the nasal glands found in hyperplastic rhinitis and leading either to destruction or to atrophy of the glands must also be due to allergy. This statement, of course, does not suggest a complete solution of the problem, for the further question arises whether the glandular hyperactivity is due to irritation from the periglandular infiltration or from nervous stimuli or to another factor. This question is certainly open to discussion, not only as it concerns the bronchial glands but also as it concerns the nasal glands. Without pretending to answer the question, one should bear in mind the fact that Maximow [16] produced similar glandular changes by dissecting the secretory nerves of the salivary glands.

SUMMARY AND CONCLUSIONS

Periglandular infiltration plays a role in the metabolism of the glands unless there is an extremely intense infiltration of lymphocytes and plasma cells or many neutrophils or eosinophils between the lymphocytes or an actual invasion of the glands by lymphocytes, the latter findings being indicative of chronic inflammation.

Under normal circumstances the nasal glands, like the salivary glands, are continuously active. As a rule, expulsion of the total amount of secretion does not take place, and, therefore, exhausted glands are seldom observed.

Onkocytes were found most frequently in old persons.

When the nasal glands and the nasal epithelium are exposed to toxins, as in purulent or syphilitic inflammation of the nasal mucous membrane, the first thing that happens is the cessation of the production

35. Kountz, W. B., and Alexander, H. L.: Death from Bronchial Asthma, Arch. Path. **5**:1003 (June) 1928.

of mucus. Later on, the glandular cells become necrotic and might be destroyed entirely by invading lymphocytes and leukocytes. Both mucous and albuminous glands are involved, although the mucous glands are involved first.

The hyperplastic mucous membrane of the nose may contain (*a*) normal glands, (*b*) hyperactive glands, with or without retention of secretion, and (*c*) atrophic glands.

The hyperactivity concerns chiefly the mucous glands and may lead to (*a*) exhaustion, (*b*) rupture of the gland or (*c*) formation of cysts. The exhaustion, in turn, leads to atrophy.

The cysts may originate in the gland tubules, in the excretory tubes or in the crypts. In the first instance the epithelium of the cyst is flattened; in the latter cases the epithelium is cuboid or even higher and the cyst may be surrounded by a thickened basement membrane.

The first evidence of the effect exerted on the glands by the edema of nasal polyps is the complete or almost complete cessation of the production of mucus in the epithelium as well as in the glands.

Furthermore, the glandular heaps, as well as the areas of periglandular infiltration, are shredded, and the individual alveoli, together with the lymphocytes and plasma cells, are scattered throughout the edematous area. Therefore, the glandular alveoli must undergo degeneration, which, in turn, may lead to complete destruction of the gland or to formation of cysts.

Purulent as well as hyperplastic inflammation of the nasal mucous membrane leads to atrophy and necrosis of the nasal glands, the first by toxins and the second by dysfunction of the glands.

The same dysfunction of glands was found by other investigators in the bronchial mucous membrane of patients who succumbed to allergic asthma and in the salivary glands after dissection of the secretory nerves.

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NASAL DEFORMITY IN ANHIDROTIC ECTODERMAL DYSPLASIA

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Among the various dysplasias, or defects, of the ectodermal appendages, Goeckermann [1] differentiated the syndrome of hereditary ectodermal dysplasia of the anhidrotic type as a definite clinical entity.[2] Early cases of this condition were described by Thurnam [3] in 1848 and by Guilford [4] in 1883. In 1938 Lord and Wolfe [5] listed about 50 cases collected from the world literature. However, most of the authors have agreed that the small number of cases reported is due not so much to the rarity of the syndrome as to the unfamiliarity of members of the medical profession with the clinical picture.

The main symptoms of this condition are dysplasias of the ectodermal appendages—the sweat glands, hair, teeth and nails—resulting in anhidrosis, hypotrichosis, anodontia and dystrophy of the nails. The other symptoms which are most consistently observed are deformity of the nose and papillary lesions of the skin. Because of the nasal deformity and protruding supraorbital ridges, most of the patients have a characteristic facial appearance (figs. 1 and 2). Frequently, ozena is also present. Other congenital malformations, seemingly unrelated to the syndrome, such as cardiac defects, malformations of the external ear, epicanthus and polydactylism, are sometimes observed in these patients.

The clinical picture of anhidrotic ectodermal dysplasia may vary considerably, depending on the degree of development of each of the component symptoms. If the anhidrosis is fully developed, it is this condition which attracts particular attention. Because of the inability to perspire, there is serious impairment in the regulation of temperature

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1. Goeckermann, W. H.: Congenital Ectodermal Defect, Arch. Dermat. & Syph. **1**:396 (April) 1920.

2. The name was subsequently suggested by Weech (Weech, A. A.: Hereditary Ectodermal Dysplasia [Congenital Ectodermal Defect]: Report of Two Cases, Am. J. Dis. Child. **37**:766 [April] 1929).

3. Thurnam, J.: Two Cases in Which the Skin, Hair, and Teeth Were Very Imperfectly Developed, Med.-Chir. Tr. **13**:171, 1848.

4. Guilford, S. H.: A Dental Anomaly, Dent. Cosmos **25**:113, 1883.

5. Lord, L. W., and Wolfe, W. D.: Hereditary Ectodermal Dysplasia of Anhidrotic Type (Congenital Ectodermal Defect), Arch. Dermat. & Syph. **38**:893 (Dec.) 1938.

and the patients affected are subject to hyperpyrexia and to severe discomfort in warm weather. However, in the majority of cases the anhidrosis is only partially developed and the patient may not be at all aware of this condition. The hypotrichosis is especially conspicuous

Fig. 1.—Photographs of patients in early cases of anhidrotic ectodermal dysplasia: *A*, Guilford's case, 1883; *B*, Goeckermann's [1] case, 1920. Note the resemblance to the patient in the case reported here (fig. 2).

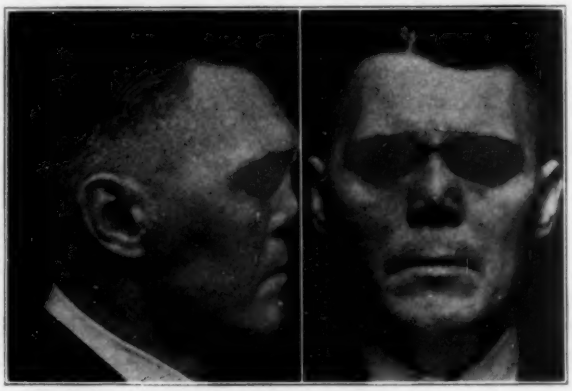

Fig. 2.—Photograph showing the facial appearance characteristic of persons with anhidrotic ectodermal dysplasia. The original contour of the nose was somewhat changed but not improved by the insertion of a bone graft twenty-three years ago. Note the protruding supraorbital ridges, the hairless facial skin (except for some hair on the upper lip and chin), the nodule between the eyebrows and the papules on the right cheek.

when it affects the eyebrows, the facial hair in males or the scalp hair in females. In some cases complete anodontia attracts the attention

of the dental surgeon (Guilford). In general, the lack of a few teeth or malformation of those in the mouth constitute the dental symptoms. In still other cases the apparently congenital ozena puzzles the otolaryngologist (Nager).[6] The nasal deformity, with or without ozena, may be the only symptom of which the patient is aware and has often led to the detection of the entire syndrome (Goeckermann).

Clouston[7] described what he called the hidrotic form of ectodermal dysplasia. Anhidrosis and nasal deformity are absent, but most of the other symptoms are identical with those of the anhidrotic form. It is not unlikely that the hidrotic form is a mild modification of the latter.

The hereditary character of anhidrotic ectodermal dysplasia has been definitely established. However, the contention of some authors that it is a sex-linked characteristic, recessive in females, is contradicted by the fact that few cases of its occurrence in females have been described in the literature.[8]

THE NASAL DEFORMITY

An external nasal deformity has been mentioned in a large majority of the reports of cases of anhidrotic ectodermal dysplasia. For the most part, it has been described briefly as a saddle back deformity. However, if one may judge from published photographs, there has usually been an abnormal shortening and general flatness of the bridge. Furthermore, as in the case to be reported presently, there is frequently a lack of forward development of the maxillary compound, characteristic of the dish face deformity.

The deformity of the nose is not specific for anhidrotic ectodermal dysplasia, and the condition can hardly be diagnosed without at least some of the other symptoms. Chondrodystrophy, congenital syphilis, trauma in infancy and other conditions may result in a similar deformity. The common etiologic factor is an arrest in growth in early postfetal life. In anhidrotic ectodermal dysplasia the nasal deformity is never noted before late childhood and seldom before the end of the first ten years of life.

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6. Nager, F. R.: Ueber das Vorkommen von Ozäna bei angeborenen Haut- und Zahnanomalien, Arch. f. Laryng. u. Rhin. **33**:426, 1920.

7. Clouston, H. R.: The Major Forms of Hereditary Dysplasia, Canad. M. A. J. **40**:1, 1939.

8. Thadani claimed this mode of inheritance for the cases of the "Bhudas of India, a toothless type of man" who does not perspire. His work and De Silva's make it seem likely that anhidrotic ectodermal dysplasia is relatively common in India (Thadani, K. I.: A Toothless Type of Man: The Bhudas of India; a Case of Sex-Linked Inheritance, J. Heredity **12**:87, 1921. De Silva, P. C. C.: Hereditary Ectodermal Dysplasia of Anhydrotic Type, Quart. J. Med. **8**:97, 1939).

Little is known about how such a nasal deformity develops. Therefore, it may be interesting to analyze this process in the light of present knowledge of the development of the nasal and facial skeleton.

Premature ossification, for example, of the epiphysial lines of the femur (located at each end of the bone) will obviously lead to an abnormal shortness of the leg. The study of the normal and pathologic growth of the facial skeleton is made difficult, however, by the presence of a large number of ossification centers. The skull consists of several independent bones which influence the facial contour directly or indirectly. Each of them has several ossification centers.[9] For instance, the sphenoid has seven pairs of such growth centers, which appear and fuse with each other at different times. Most of the centers become fused shortly after birth, thus completing the endochondral ossification. Further growth of the facial skeleton occurs by apposition of new bony layers.

Virchow[10] pointed out that the synchondrosis spheno-occipitalis behaves differently, because normally the ossification there is not completed before adolescence. He attributes the normal forward growth of the facial skeleton (specifically of the maxillary compound and the nose) to properly timed ossification between the occipital and sphenoid bones. When ossification is completed prematurely, long before the facial contour is established, the clivus sphenoidalis remains short and vertical and there is a lack of forward development of the maxilla. The maxillary compound not only forms the foundation of the nose but is an integral part of its structure. The elevation of the nasal bridge is due more to the development of the frontal processes of the maxilla than to the size of the nasal bones.

Ossification of the perpendicular plate of the ethmoid bone and of the vomer occur similarly in postfetal life. The development of these structures largely determines the contour of the nose. The ossification of the perpendicular plate achieves its final stage between the third and the fifth years, at which time the plate fuses with the vomer, but ossification of the latter is completed only before puberty.[11] The parts of the ethmoid bone which constitute the lateral walls of the nasal cavities are also not fully developed before puberty.

In normal growth, the endochondral ossification of the skull continues in postfetal life only in the synchondrosis spheno-occipitalis and in

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9. Spalteholz, W.: Handatlas der Anatomie des Menschen, ed. 6, Leipzig, S. Hirzel, 1919, vol. 1, pp. 65-70.

10. Virchow, R.: Ueber einige merkmale niederer Menschenrassen am Schädel, Berlin, F. Dümmler, 1875.

11. Carter, W. W.: Clinical Observations of the Influence of the Nasal Septum and the Development of the Nose and the Palatal Arch, Laryngoscope **45**:355, 1935.

the bones which form the nose. If for some unknown reason this growth is halted in early childhood and does not continue into adolescence, a facial deformity, such as that which occurs in anhidrotic ectodermal dysplasia, will develop. Underdevelopment of the lateral parts of the ethmoid bone will result in abnormally wide nasal cavities.

The most active vertical growth of the facial skeleton occurs between the ages of 5 and 7, owing to the development of the alveolar processes and the teeth in the upper and lower jaws. This may have a bearing on the character of the nasal deformity in anhidrotic ectodermal dysplasia, since in cases of anodontia the vertical growth of the face is less pronounced and the shortness of the nose is not so accentuated as in cases in which the dental development is normal.

It is difficult to explain the deformity of the facial skeleton in connection with a syndrome involving the ectodermal appendages. Therefore, the hypothesis presents itself that the primary defect lies in the anterior lobe of the hypophysis, which is of ectodermal origin and which exercises some influence on skeletal growth. No gross pathologic condition of the hypophysis has ever been reported in connection with anhidrotic ectodermal dysplasia, but there have been several observations regarding the smallness of the sella turcica in such cases. It is noteworthy that no abnormality of skeletal growth has been observed other than that associated with the facial development.

OZENA

Ozena, or atrophic rhinitis, is a condition well known to rhinologists and characterized by the putrid odor, formation of crusts, atrophy of the mucous membrane and spaciousness of the nasal cavity. However, most authors believe that its causation is not uniform.

Steiner [12] differentiated between two kinds of ozena: (1) Gruenwald's form, which is a sequel to a chronic infection causing atrophy of the mucosa as well as of the underlying bony structures, and (2) Fränkel's form, which is due to a congenital malformation of the nasal cavities. The wideness of the nasal cavities and the lack of full development of the lateral structures are responsible for the consequent chronic infection and atrophy of the mucous membrane.

Congenital ozena develops in childhood and puberty, whereas the acquired condition is a disease of maturity. Steiner described what he called the "ozena face," characterized by a flat or saddle back short

12. Steiner, M.: Zur Weiterentwickelung der Lehre von der Ozaene, Arch. f. Laryng. u. Rhin. **21**:282, 1908.

nose and protruding forehead and bearing a striking resemblance to the facial appearance noted in cases of anhidrotic ectodermal dysplasia.

Ozena is a frequent symptom of anhidrotic ectodermal dysplasia (Nager's case).[13] In view of the previously discussed abnormal development of the nose, it is clear that the presence or absence of ozena will depend on the character and extent of the nasal malformation. When the pathologic process leads to the formation of abnormally wide nasal passages, the basis for Fränkel's form of ozena is present.

REPORT OF A CASE

A 45 year old patient of German descent was referred to me in February 1940 for correction of a nasal deformity which had been repeatedly and unanimously diagnosed as congenitally syphilitic.

History.—The deformity had been present as long as the patient could recall, but it became more pronounced at the beginning of puberty. No pertinent family history could be obtained, because since childhood the patient had had no contact with his relatives, all of whom lived abroad. He knew that, after his father's death, his mother had married a close relative of hers, and he remembered having been told that his stepfather had a nasal deformity similar to his own.

The patient had always been in good health. He was married and had a normal child, who died in her early adolescence after an appendectomy.

The patient was sensitive about his appearance. He was shy and withdrawn, and both he and his wife avoided social contacts. He worked as a chef and was recognized for his skill. However, each time when he had to change his position, he encountered a hostile and suspicious attitude and had great difficulty in obtaining a satisfactory job. Innumerable times he was requested to have his blood examined. It was always normal.

The patient volunteered the information that he had noticed that he perspired little, particularly above his waist. He was rather proud of the fact that during hot seasons, while his working companions in the kitchen were constantly mopping their faces, he remained dry and clean.

In 1917 (at the age of 22) he sought medical attention, to improve his appearance, and was operated on. A fragment of his own rib was inserted into his nose. This procedure brought no improvement, and he has been complaining since of "rheumatism" at the donor site in his chest.

Findings.—The patient was of average size and weight but of rather delicate and fragile build. His intelligence was normal. His voice was relatively high pitched. His face (fig. 2) had a striking, almost repulsive bulldog appearance. The lower portion of his forehead and the supraorbital ridges bulged, whereas the middle portion of his face receded. The root of his nose seemed to be pulled in; the nasal bridge was extremely flat and short, thus exposing an objectionably wide, somewhat receding upper lip. The nasal tip was pulled down.

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13. Christ, J.: Nase und Ohre bei angeborenen Mangel der Schweissdrüsen. Ein Beitrag zur Ozänafrage, Ztschr. f. Laryng. u. Rhin. **6**:391, 1913.

Anterior rhinoscopy revealed that the cartilaginous septum was soft and wrinkled, offering no support to the bridge. Posteriorly, the perpendicular plate appeared to be present and to be straight. The turbinates were poorly developed. At the junction with the vestibulum, the nasal cavities were narrowed by folds of contracted mucous membrane. No crusts or discharge was present.

The patient had an upper denture, replacing five teeth (allegedly extracted a few years ago). The remaining teeth were all present and except for several fillings appeared to be normal in shape and form.

The skin on the face was smooth, dry and devoid of hair with the exception of a few patches on the upper lip and under the chin. There was a soft fibromatous mole in the glabular region. Several small, slightly elevated papules spread over the cheek. The eyebrows were present. The hair on the scalp was thin and dry and had a brittle quality.

The skin over the body was strikingly white and smooth and free of hair, with the exception of some blond hair in the axillas and on the pubes. There was a yellow, slightly elevated mole on the left side of the chest.

The nails on both hands were thickened, short and unevenly broken off, with multiple cracks and grooves.

In other respects, the physical examination revealed nothing abnormal. The Wassermann reaction was negative.

Comment.—The characteristic facial appearance resulting from the protruding forehead and the undersized nose, the hypotrichosis (particularly conspicuous on the patient's face), the hypohidrosis, the dystrophy of the nails and the numerous papillary lesions of the skin leave no doubt that the case was one of anhidrotic ectodermal dysplasia. In this case the condition can certainly be considered of mild character, because the anhidrosis was not fully developed and did not cause the patient any noticeable discomfort. When the patient was on the operating table, I had the opportunity to observe that he perspired, but much less than an average person would. A further investigation of the degree of hypohidrosis and the distribution of the sweat glands was not possible, because the patient was not concerned about this and refused to undergo any tests. No malformation of the teeth was noted, and the lack of five teeth must be attributed to an extraction for some independent reason. A biopsy of skin from the neck showed definite atrophy of the epidermis, scarcity of sudoriparous glands and some focal degenerative changes in the sebaceous glands. The cutaneous papules were of nevus-like structure.

SURGICAL MANAGEMENT OF THE NASAL DEFORMITY IN ANHIDROTIC ECTODERMAL DYSPLASIA

Although a nasal deformity is present in most cases of anhidrotic ectodermal dysplasia, surgical correction is mentioned in the literature only once (Goeckermann). Undoubtedly, several patients with this

Fig. 3.—The final result. Note the change in facial expression. The result was extremely gratifying, not only because of the physical change but because of the improvement in the patient's mental attitude.

The reconstructive procedure consisted of seven major and minor operations, spaced over a period of eight months. The total time of hospitalization was nine days, and the patient was out of work for about three weeks.

Fig. 4.—First stage of reconstruction. The tip of the nose was severed and pulled down to the desired position. In this manner, lengthening of the nose is achieved through reconstruction of the middle portion. A flap on the neck was prepared; its medial part, which was to serve as a pedicle, was tubed, and its lateral part, which was to be used for reconstruction, was delayed. The use of the neck flap was possible in this case because this skin was not hair bearing. A classic forehead flap would have been simpler, but the patient refused to have his forehead scarred. The skin over the proximal part of the nose was delayed, to serve as a lining.

condition have undergone plastic operations, most of them after a diagnosis of syphilitic deformity. In mild cases the patient may lead a normal life and engage in normal activities, but there is an under-

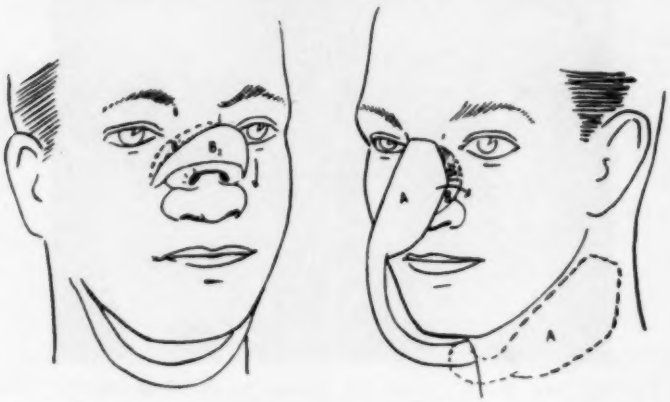

Fig. 5.—Diagram illustrating the procedure used. The delayed flap (*B*) was turned down 180 degrees and sutured to the detached nasal tip, in order to serve as a lining. The pedicled flap (*A*) was used to serve as a skin cover for the reconstructed portion as well as for the donor site of flap *B*.

Fig. 6.—Second stage of reconstruction. The pedicled flap remained in this position for three weeks. The patient was comfortable and able to move his head freely. He wore a light dressing but continued to work.

standable need for rehabilitation, both because of the patient's repulsive appearance and because of the social stigma of syphilis which is associated with it.

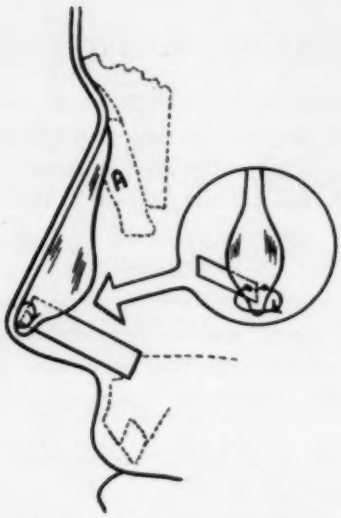

Fig. 7.—Diagram showing the arrangement of the two cartilage grafts which were used for reconstruction. (*A* represents the old bone graft, which was left undisturbed.) Before the cartilage grafts were inserted, it was necessary to thin down the lateral parts of the flap. Preserved cadaver cartilage (stored according to Pierce and O'Connor's [14] method) was used.

Fig. 8.—Roentgenogram of the nose after reconstruction. The bone graft is plainly visible, but the cartilaginous grafts can be recognized only by their outline. Note the receding maxilla.

14. O'Connor, G. B., and Pierce, G. W.: Refrigerated Cartilage Isografts. Surg., Gynec. & Obst. **67**:796, 1938.

The surgical procedure for correction of the nasal deformity in anhidrotic ectodermal dysplasia varies, depending on the morphologic analysis of the deformity. For the sake of brevity, the surgical management of the case reported here is presented through photographs and diagrams.

SUMMARY

An "undersized" nasal deformity is a consistent symptom of anhidrotic ectodermal dysplasia and is responsible for the characteristic facial appearance of patients with this condition. Other symptoms are: anhidrosis, hypotrichosis, anodontia, dystrophy of the nails and papillary lesions of the skin.

The facial deformity in anhidrotic ectodermal dysplasia is due to an arrest in early childhood of the endochondral ossification of portions of the skull, a process which normally continues into adolescence.

The indication for surgical reconstruction is the patient's repulsive appearance and the stigma of syphilis commonly associated with it.

A case of anhidrotic ectodermal dysplasia is reported, and the surgical management of the nasal deformity is presented diagrammatically.

Dr. Rudolph Singer assisted in making the diagnosis.

TUBERCULOSIS OF THE EAR

BRUCE PROCTOR, M.D.
DETROIT
AND
J. R. LINDSAY, M.D.
CHICAGO

Tuberculosis of the temporal bone was described as far back as the early eighteenth century, by Jean Louis Petit.[1] In the first half of the nineteenth century the teaching of Laennec with respect to pulmonary tuberculosis led other workers to study the tuberculous changes in the temporal bone in patients with this disease. Eschle [2] first identified the tubercle bacillus in a lesion of the ear in 1883, only one year after Koch's discovery of that organism. In 1885 Habermann [3] first demonstrated tuberculous lesions in the tympanic mucous membrane. In the eighteen nineties the routes of infection were studied theoretically and clinically. The theory that the eustachian tube was the portal of entry was favored by some,[4] while others favored the idea that infection was carried by the hematogenous route.[5] In 1913 Brieger [4a] attempted to classify tuberculous otitis media as lupoid, infiltrating, fungoid or necrotizing. In 1922 Leegaard [6] expressed disapproval of Brieger's classification and insisted that the disease is universal in character. In 1926 [7] the direct relationship of aural tuberculosis to the presence of tuberculosis elsewhere in the body was especially emphasized by Cemach.

From the Division of Otolaryngology, Department of Surgery, the University of Chicago.

1. Petit, J. L., cited by Cemach.[7b]

2. Eschle: Tuberkelbacillen in dem Ausflusse bei Mittelohreiterungen von Phthisikern, Deutsche med. Wchnschr. **9**:441, 1883.

3. Habermann, J.: Ueber die tuberculöse Infection des Mittelohrs, Ztschr. f. Heilk. **6**:367, 1885.

4. (*a*) Brieger, O.: Die Tuberkulose des Mittelohres, Verhandl. d. deutsch. otol. Gesellsch. **22**:31, 1913. (*b*) Bondy, G.: Zur Pathogenese der Mittelohrtuberkulose, Monatschr. f. Ohrenh. **43**:24, 1909. (*c*) Gradenigo, G.: Lupus des mitteleren und inneren Ohres, Allg. wien. med. Ztg. **33**:399, 1888.

5. (*a*) Barnick, O.: Klinische und pathologisch-anatomische Beiträge zur Tuberculose des mitteleren und inneren Ohres, Arch. f. Ohrenh. **40**:81, 1896. (*b*) Preysing, H.: Multiple tuberkulöse Tumoren am Schädel und in beiden Trommelfellen, Ztschr. f. Ohrenh. **32**:369, 1898.

6. Leegaard, F.: Beitrag zum Studium der Mittelohrtuberkulose, Beitr. z. prakt. u. theoret. Hals-, Nasen- u. Ohrenh. **18**:135, 1922.

7. Cemach, A. J.; (*a*) in Denker, A., and Kahler, O.: Handbuch der Hals-Nasen-Ohrenheilkunde, Berlin, Julius Springer, 1926, vol. 7, p. 590-649; (*b*) Das Problem der Mittelohrtuberkulose, Berlin, Urban & Schwarzenberg, 1926.

INCIDENCE OF TUBERCULOUS OTITIS MEDIA

The incidence of clinically recognizable tuberculous otitis media as compared with the incidence of the nonspecific forms of otitis media has been estimated by several authors.[8] They found that in 1.3 to 15.4 per cent of the cases of otitis media the condition was due to tuberculosis. By combing their results one finds that out of a total of 8.555 cases of otitis media the average incidence of tuberculous otitis media was 2.7 per cent. This wide variation in percentage is probably due to the difference in the local incidence of tuberculosis in various parts of the world and also to the various technics employed in establishing the diagnosis.

Other workers studied the incidence of clinically recognizable tuberculous otitis media in patients with pulmonary tuberculosis. They found the incidence to vary from 1 to 3 per cent.[9]

The incidence of chronic suppurative otitis media which has not proved to be tuberculous in patients with pulmonary tuberculosis has been reported as follows: Rüedi,[9d] 10 per cent; Krymon,[10] 11 per cent; Cemach,[7] 11 per cent; Brüggemann,[9e] 9.8 per cent; Lussmann and Bendove,[11] 7.5 per cent; Miller,[9b] 4.7 per cent. It has been generally assumed that such cases were examples of nonspecific chronic otitis media, but in most of these cases tuberculosis had not been definitely excluded.

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8. Leegaard, F.: Tuberculosis of the Middle Ear, Laryngoscope **31**:374, 1921. Guthrie, D.: Notes on Thirteen Cases of Aural Tuberculosis in Infants, J. Laryng. & Otol. **35**:99, 1920. Souper, H. R.: Tuberculous Disease of the Middle Ear and Mastoid, Edinburgh M. J. **41**:154, 1934. Voss, O.: Speziellen Chirurgie des Ohres, ed. 3, 1925, vol. 2. Assheuer, P.: Die Mittelohrtuberkulose, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:322, 1933. Cox, G. H., and Dwyer, J. G.: Tuberculosis of the Middle Ear, Arch. Otolaryng. **9**:414 (April) 1929; Final Report on the Isolation and Cultivation of the Tubercle Bacillus from the Discharging Ear in Cases of Chronic Purulent Otitis Media, J. Laryng. & Otol. **31**:288, 1916. Turner, A. L., and Fraser, J. S.: Tuberculosis of the Middle Ear Cleft in Children: A Clinical and Pathological Study, ibid. **30**:209, 1915. M'Cart, H. W. D.: Tuberculous Disease of the Middle Ear, ibid. **40**:456, 1925. Leegaard.[6] Cemach.[7]

9 (*a*) Thomson, St. C.: Tuberculosis of the Middle Ear as Met with in Adults in a Sanatorium, J. Laryng. & Otol. **46**:449, 1931. (*b*) Miller, J.: Aural Tuberculosis, Arch. Otolaryng. **20**:677 (Nov.), 1934. (*c*) Ormerod, F. C.: Tuberculous Disease of the Middle Ear, J. Laryng. & Otol. **46**:449, 1931. (*d*) Rüedi, L.: Zur Klinik und Therapie des Mittelohrcholeasteatoms, Schweiz. med. Wchnschr. **64**:411, 1934. (*e*) Brüggemann, A.: Die Ohrtuberkulose, Ergebn. d. ges. Tuberk.-Forsch. **9**:109, 1939. (*f*) Cemach.[7]

10. Krymon, A.: Tuberkulose des Mittelohres. Bakteriologische und klinische Untersuchungen, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **18**:108, 1931.

11. Lussmann, F. J., and Bendove, R. A.: Chronic Otitis Media in the Tuberculous, Arch. Otolaryng. **6**:153 (Aug.) 1927.

PATHOGENESIS OF AURAL TUBERCULOSIS

It is known that nearly all persons become infected with tuberculosis in infancy or childhood. The initial infection usually occurs in the lungs except in regions where milk-borne tuberculosis is still prevalent. In tuberculosis of bovine origin the initial lesion may occur in the intestinal tract or at times in the pharyngeal lymphatic tissues.

When the initial lesion appears in a lung, it is characterized by the formation of a small subpleural tubercle (Ghon tubercle). This is usually termed "the primary effect." During the development of this initial lesion the lymphatics become infected, and regional tuberculous lymphadenitis results. The combination of the primary lesion and the secondary lymphadenitis is called "the primary complex." This primary complex usually becomes quiescent and heals, leaving a calcified primary lesion, which is often observed at necropsy. However, the primary complex may not heal. In such an event the tubercle bacilli may continue to spread through the lymph channels and eventually may infect the blood stream, with the result that multiple hematogenous foci of tuberculosis are produced. This process may run an acute or a chronic course. In chronic cases, after the primary complex eventually becomes encapsulated or partially calcified, it is possible for dissemination by the blood stream to occur from time to time. It is only by such transitory tubercle bacillemia that one can explain the isolated metastatic lesions, which are remote from the natural portals of entry of the tubercle bacillus. These metastatic lesions are especially likely to become problems of the surgeon. There is no doubt that in some cases so-called primary tuberculosis of the temporal bone has this origin. Most of the reported cases of primary tuberculosis of the temporal bone have not been studied completely, either in life or at necropsy, to determine the presence or absence of healed or active tuberculous foci elsewhere in the body. It should be emphasized that in the majority of these cases the disease in the temporal bone is primary in a clinical and not in a pathologic sense. It is always problematic whether aural tuberculosis is ever primary, as Cemach [7] has suggested. At least, the direct entrance of tubercle bacilli into the ear through the eustachian tube or through a perforated drum in the absence of tuberculosis elsewhere in the body must be extremely rare.

Obviously, the ultimate results of a hematogenous dissemination to the temporal bone or to any other structure in the body are dependent on a number of factors, such as the degree of activity of the original focus, the general resistance of the patient and the local resistance of the structures involved by the dissemination.

Tuberculosis of the temporal bone has been produced experimentally in animals by the injection of suspensions of tubercle bacilli into the

common carotid artery.[12] Theissing was able to demonstrate miliary tubercles in the bone marrow of the temporal bone and to a lesser extent in the mucous membrane of the tympanum. In some cases lesions in the mucous membrane were absent. Similar lesions in the bone marrow and mucous membranes of human temporal bones have been reported by others.[13]

Many authors have expressed the belief that the eustachian tube is an important portal of entry for tubercle bacilli giving rise to otitis media in patients with pulmonary tuberculosis and in infants. It is thought that during coughing and vomiting infected sputum may reach the tubal orifices and eventually may gain access to the eustachian tube and the middle ear. In those cases, however, in which complete post-mortem studies, with histologic examination of the temporal bones and eustachian tubes, have been made, all the evidence obtained has favored the theory that infection takes place by the hematogenous route. When advanced destruction by the disease has occurred, it is usually no longer possible by histologic examination to determine the route of infection. On the other hand, a histologic examination made relatively early in the course of tuberculosis of the middle ear provides definite evidence of a hematogenous origin.

Our findings in a number of cases of tuberculosis of the temporal bone in an early stage provide strong evidence that the common pathway by which tubercle bacilli reach the ear is the hematogenous one.

REPORT OF CASES

CASE 1.—M. M., aged 4 years, came to the University of Chicago Clinics on Sept. 8, 1932 with a history of headache, vomiting, loss of weight and fever of a week's duration. Her stepfather had active pulmonary tuberculosis. The tuberculin test the following day was positive. Roentgenograms of the chest revealed dense areas of soft tissue in the upper and middle portions of the hilus of the right lung. A diagnosis of tuberculous meningitis was made and was substantiated by studies of the spinal fluid. The child gradually became worse and died twenty-six days after admission.

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12. Theissing, G.: Pathoiogisch-anatomische und experimentelle Untersuchungen zur Pathogenese der Mittelohrtuberkulose, Ztschr. f. Laryng., Rhin., Otol. **22**:328, 1932.

13. (*a*) Henrici: Weitere Erfahrungen über die Tuberculose des Warzen-fortsatzes im Kindesalter, Ztschr. f. Ohrenh. **51**:125, 1906. (*b*) Ghon, A., and Roman, B., cited by Brüggemann.[9e] (*c*) Grünberg, K.: Zur Frage der hämatogenen Osteomyelitis des Schläfenbeines, Ztschr. f. Hals-, Nasen- u. Ohrenh. **21**:192, 1928. (*d*) Richter, H.: Beiträge zur pathologischen Anatomie des Ohres, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **141-142**:167, 1936. (*e*) Zarfl, M.: Ueber primäre Mittelohrtuberkulose beim Säugling, Virchows Arch. f. path. Anat. **266**:274, 1927. (*f*) Altmann, F.: Ein Beitrag zur Frage der primären Mittelohrtuberkulose, Monatschr. f. Ohrenh. **65**:460, 1931. (*g*) Kleinschmidt, H., and Schürman, P.: Primére Mittelohrtuberkulose im frühen Säuglingsalter, Monatschr. f. Kinderh. **40**:193, 1928. (*h*) Barnick.[5a] (*i*) Theissing.[12]

Autopsy.—The postmortem observations were as follows: large caseonodular tubercles in the right lung and hilar lymph nodes (unhealed primary complex), with lymphogenous extension to the mediastinal and abdominal lymph nodes; acute generalized hematogenous miliary tuberculosis; acute exudative tuberculous basilar leptomeningitis, and disseminated miliary and small fibrocaseous tubercles in the liver, spleen, bone marrow, lungs, leptomeninges, kidneys and ileum.

Histologic Examination of One Temporal Bone (side not recorded).—The air cell spaces of the middle ear were normal throughout. The petrosa was composed of young marrow. Several foci of fibrocaseous miliary tuberculosis were present. In the internal auditory meatus was an acute exudative infiltration of the leptomeninges. Occasional giant cells were present. In the internal ear was evidence of labyrinthitis in an early stage—most marked near the cribriform plate.

Clinically and pathologically case 1 illustrates the form of tuberculosis in which overwhelming infection or poor resistance favors progression rather than healing of the primary complex. A lymphogenous spread from the large primary focus led to extensive caseous necrosis of mediastinal and abdominal lymph nodes. From these nodes bacilli must have entered the blood stream via the veins or the thoracic duct. There probably were successive episodes of tuberculous bacillemia, since the metastatic miliary lesions were of different sizes and presumably of different ages. As is so often the case in generalized miliary tuberculosis, death was the result of tuberculous meningitis. There was the usual concentration of small metastatic tubercles in the liver, spleen and bone marrow—the important filter beds of the reticuloendothelial system. Several small foci of fibrocaseous tuberculosis were present in the bone marrow of the petrosa. There was no evidence of tuberculosis in the air cell system of the middle ear.

This case illustrates the localization of tubercle bacilli in the bone marrow of the temporal bone as a part of generalized, quickly fatal hematogenous miliary tuberculosis. A disease process of this type, without recognized aural symptoms, must be extremely common in persons who die of an overwhelming tuberculous bacillemia. The importance of a case of this type is that it focuses attention on the hematogenous route of infection of the ear by tubercle bacilli.

Case 2.—A. M., 22 months of age, was admitted to the University of Chicago Clinics on Dec. 7, 1938 because of a cough of three months' duration and the presence of fever, with irritability, for three weeks.

Examination revealed dyspnea, rales in the chest and harsh breath sounds. The white blood cell count was 25,000, with 48 per cent polymorphonuclear cells. The tuberculin test was positive. Roentgenograms of the chest made the following day revealed a widespread involvement of the lung fields, associated with an enlargement in the upper mediastinum, particularly on the right. A diagnosis of miliary tuberculosis was made. Further roentgen studies five days later showed an increased dissemination of tuberculous lesions in the lung fields.

Thirty-five days after admission the child became listless. Studies of the spinal fluid at this time revealed a slight increase in pressure; the fluid was clear, with

1 plus globulin and 26 mg. of sugar per hundred cubic centimeters; the cell count was 6; no organisms were demonstrable on smear or guinea pig inoculation. Forty-two days after admission the respirations became irregular, the neck became moderately rigid, the Kernig test was positive and the patient died.

Autopsy.—The observations at autopsy were: large soft caseonodular tubercles in the left lung and hilar lymph nodes (unhealed primary complex), with lymphogenous extension to the mediastinal and abdominal lymph nodes; acute and chronic generalized hematogenous miliary tuberculosis; acute exudative and fibroplastic tuberculous basilar leptomeningitis; disseminated miliary and small fibroplastic tubercles in the liver, spleen, bone marrow, lungs, leptomeninges and kidneys; intimal tubercles in a large pulmonary vein; focal caseoulcerative tuberculous

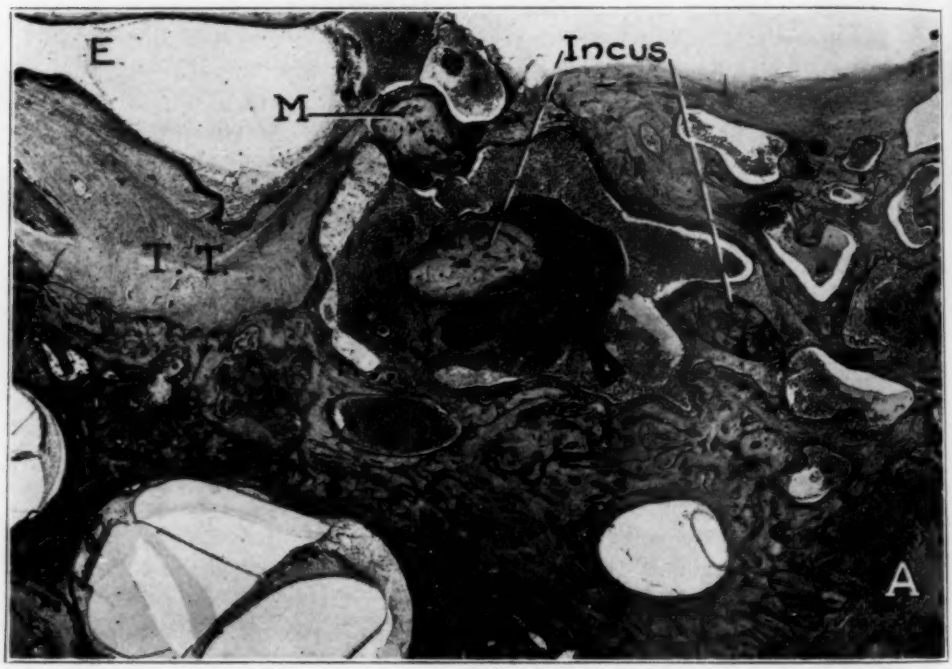

Fig. 1 (case 2).—Horizontal section (× 13) from the right ear at the junction of the epitympanum and tympanum. The patient had acute tuberculous otitis media in an early stage. The incus is embedded in granulation tissue, which also fills the antrum (*A*). Air spaces in the epitympanum and mastoid are filled with tuberculous exudate. The malleus (*M*), the tensor tympani muscle and tendon (*TT*) and the mouth of the eustachian tube (*E*) are shown.

bronchiolitis; tuberculous bronchopneumonia, with disseminated fibrocaseous miliary tubercles (bronchiogenic or acinonodose type), and small tubercles and superficial ulcers in Peyer's patches of the ileum.

Microscopic Examination.—(*a*) Right ear (figs. 1 and 2): Examination of the air cell system of the middle ear revealed that the epitympanum, the mastoid antrum and a large portion of the mastoid air cells contained considerable tuber-

culous granulation tissue. This was most marked in the region of the ossicular chain. The granulations were relatively young, with large dilated vascular channels. Single and conglomerate tubercles were scattered about. In some areas early caseation was evident. In some places the epithelium over the granulation tissue had become ulcerated, with the resultant formation of a tuberculous exudate. The eustachian tube, the hypotympanum, the secondary tympanic membrane and the stapes footplate all were normal.

The petrosa contained numerous foci of fibrocaseous miliary tuberculosis scattered in the marrow.

The internal auditory meatus contained many lymphocytes and large mononuclear cells in the leptomeninges along the nerve trunks.

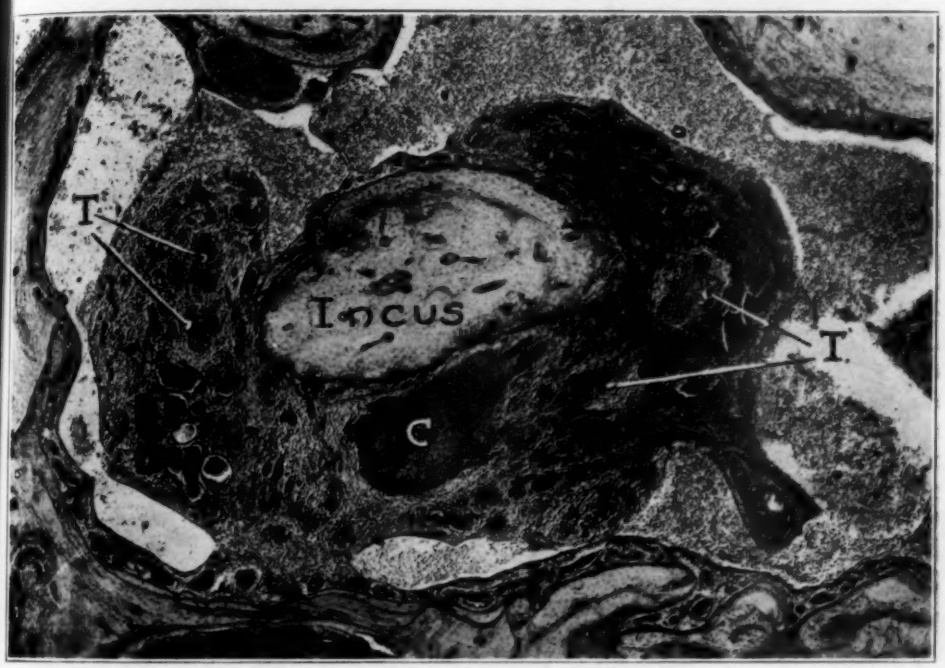

Fig. 2 (case 2).—Higher magnification (× 20) of the granulation tissue surrounding the incus shown in figure 1. Note epithelioid cell miliary tubercles (*T*), a caseous tubercle (*C*), numerous large blood vessels, and cellular exudate in the surrounding air space.

The internal ear contained mild serous exudate, in which there were a few lymphocytes. This was most marked in the regions adjacent to the cribriform plate.

(*b*) Left ear (fig. 3): On examination of the air cell system of the middle ear early inflammatory changes of a tuberculous nature were noted in the region of the ossicular chain and in a small area of the epitympanum. The granulation tissue contained many young blood channels and scattered tubercles, some of which had ulcerated and had discharged tuberculous elements into the tympanic cavity. Early caseation was present in several portions of the granulation tissue.

The mucosa of the remaining portions of the air cell system of the middle ear was moderately edematous. Tuberculous exudate was scattered throughout the air cell system. The tympanic membrane, round window membrane and stapes footplate were intact. The eustachian tube was free of disease.

The petrosa contained young cellular marrow with numerous discrete fibrocaseous miliary tubercles.

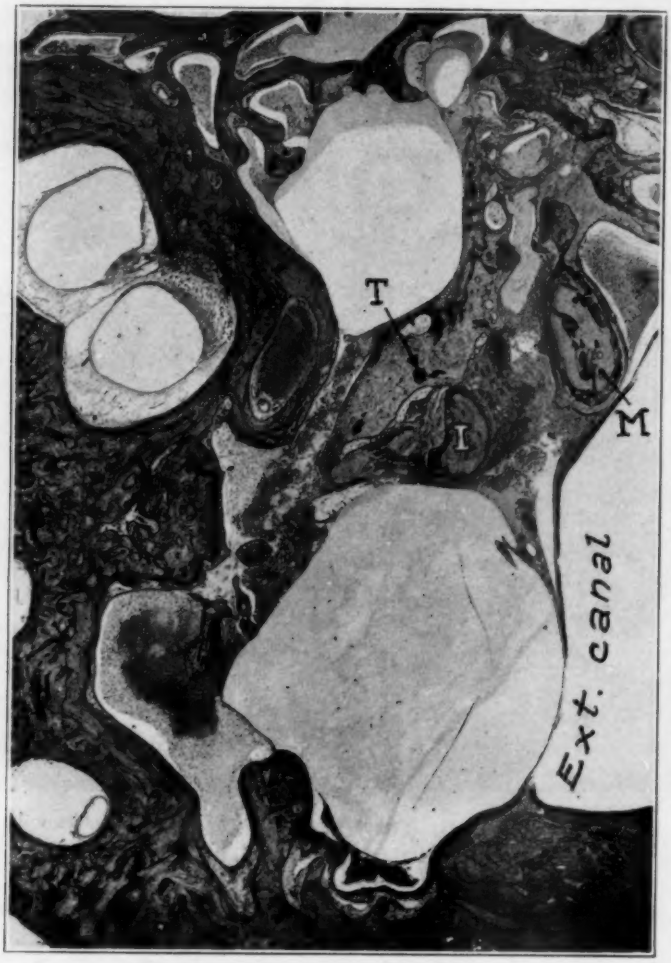

Fig. 3 (case 2).—Vertical section (× 13) from the left ear of a patient who had acute tuberculous otitis media in an early stage and miliary tuberculosis. Adjacent to the incus (*I*) is a mass of granulation tissue containing a single miliary tubercle (*T*). The air spaces have slightly thickened membranes and contain a serous exudate, which is especially dense about the ossicles. The exudate contains a few polymorphonuclear neutrophils and many large mononuclear cells. The hypotympanum and the mouth of the eustachian tube were practically free of exudate. *M* indicates the malleus.

The internal auditory meatus showed evidence of acute exudative tuberculous leptomeningitis.

The internal ear contained a slight serous exudate, with an occasional lymphocyte in regions adjacent to the cribriform plate.

The clinical and pathologic background in this case was similar to that in case 1. This child also had metastatic tubercles of different ages, as estimated by size and fibroplastic reaction. One important type of change was seen in this case which was not seen in case 1, namely, the occurrence of ulcerative and pneumonic lesions in the lungs and ulcerative lesions in the intestines. The significance of these changes is that they indicate not only chronicity but also the establishment of the adult type or allergic phase of tuberculosis. In this phase the formation of tuberculous granulation tissue, ulceration and exudation are phenomena of major importance. This change is reflected in the lesion of the ear.

It is likely that the initial localization of tuberculosis occurred in the bone marrow, although an occasional hematogenous tubercle may have developed in the mucous membrane. It is impossible to tell how many of the tubercles found in the marrow arose from bacilli which arrived in the original hematogenous dissemination and how many arose by dissemination from contiguous active foci through lymph channels. It is obvious that either a primary (hematogenous) or a secondary (from foci in bone marrow) infection of the mucous membranes has occurred. This resulted not only in tubercle formation but in exudation, ulceration and the formation of tuberculous granulation tissue. The spread of exudate containing tubercle bacilli throughout the mucosa-lined air cell system eventually led to extensive tuberculosis of the mucous membranes entirely analogous to that in bronchial walls from the spread of sputum containing tubercle bacilli. The effects were maximal in the epitympanum and aditus. The absence of clinical symptoms referable to the aural lesions must be attributed to the youth of the patient or possibly to clouding of her sensorium.

Case 3.—R. M., aged 2½ years, was admitted to the University of Chicago Clinics on May 25, 1939 in a state of coma. Bilateral otalgia had been present for eight weeks, and bilateral spontaneous otorrhea for three weeks, following an acute infection of the upper respiratory tract. Drowsiness, vomiting and a daily elevation of temperature to 102 F. had been present for a week. No history of tuberculous contact was elicited.

Physical examination revealed bilateral blurring of the optic disks, bilateral weakness of the lateral rectus muscle, slight stiffness of the neck, a bilateral positive Babinski sign and a positive Macewen's sign. The impression was bilateral mastoiditis and otitic hydrocephalus. The white blood cell count was 9,350. Roentgen examination showed bilateral mastoiditis. Examination of the spinal fluid revealed an initial pressure of 350 mm. of water, clear fluid, 1 plus globulin and a cell count of 65, with 63 per cent lymphocytes.

On the day of admission a bilateral mastoidectomy was performed. Pus and granulation tissue were evacuated, and the dura was exposed and found uninvolved. The lateral sinuses were normal. Postoperatively the child was critically ill, with marked Cheyne-Stokes respiration and attacks of generalized convulsions. Internal strabismus was marked on the left. On the second day the administration of sulfanilamide was started. On the fourth day sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) was substituted. At this time examination of the spinal fluid revealed a pressure of 600 mm. of water, clear fluid, 1 plus globulin and 22 cells, chiefly lymphocytes. The white blood cell count was 10,000. On the fifth day the tuberculin test (1:1,000) was negative. At this time the right temporal lobe was explored for an abscess and none was found. Ventriculograms

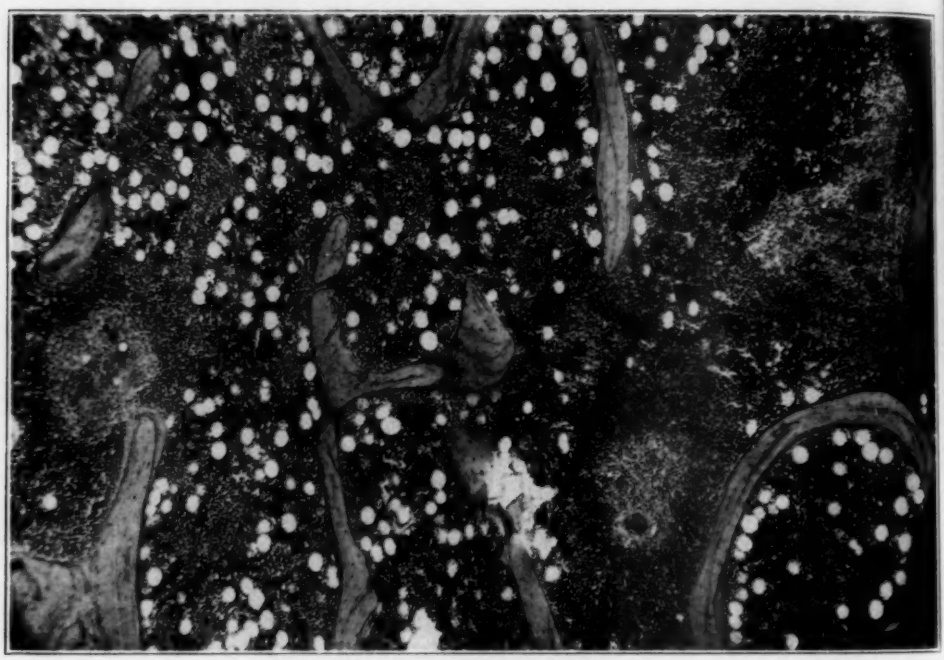

Fig. 4 (case 3).—Section (× 55) showing hematogenous tubercles in the bone marrow of the petrosa in miliary tuberculosis. Three typical fibroplastic tubercles, each containing one or more giant cells of the Langhans type are seen.

revealed no abnormalities. A Frazier needle was inserted for drainage of the cerebrospinal fluid. This ventricular fluid contained 3,000 cells, chiefly lymphocytes; 2 plus globulin; 18.8 Gm. of total proteins per hundred cubic centimeters; 632 mg. of chloride (108.4 mg. as sodium chloride), and 54.8 mg. of sugar. Culture of material from the left ear revealed Staphylococcus aureus.

The postoperative impression was acute serous arachnoiditis. Postoperatively the temperature rose to 105 F. and remained high. Large quantities of cerebrospinal fluid drained from the needle. The patient did not improve and remained in coma, with irregular respirations. He died on the eleventh day.

Autopsy.—The conditions observed at autopsy were: large soft caseonodular tubercles in the left lung and hilar lymph nodes (unhealed primary complex), with lymphogenous extension to the mediastinal and abdominal lymph nodes; acute and chronic hematogenous miliary tuberculosis; acute exudative and focal chronic proliferative basilar tuberculous leptomeningitis; internal hydrocephalus; disseminated (fresh) miliary tubercles, and miliary and conglomerate fibroplastic tubercles in the liver, spleen, bone marrow, lungs, leptomeninges and kidneys; acute ulcerative tuberculous bronchitis and acute bronchopneumonia (tuberculous?); bilateral tuberculosis of the temporal bone; freshly sutured mastoidectomy wounds, and a parietal trephine wound on the right.

Fig. 5 (case 3).—Section (× 55) from the right ear of a patient who had tuberculous mastoiditis, showing various stages of the disease. Four adjacent air cells are seen. The mucosa is greatly infiltrated and thickened. Cell 1 has intact epithelium with adherent exudate; cell 2 shows focal caseation, ulceration and exudation; cell 3, diffuse caseation, ulceration and exudation, and cell 4, a more advanced stage, with organization of the exudate. Note the central caseous debris surrounded by giant and epithelioid cells.

Microscopic Examination.—(*a*) Right ear (figs. 4, 5, 6 and 7): In the external canal a purulent exudate was present. Beneath the epithelium of the external canal were a few small tubercles containing giant cells.

The tympanic membrane was perforated. There was a marked submucosal and subepithelial infiltration by wandering cells. Giant cells were scattered beneath the mucosal epithelium. The mucosa on the tympanic surface of the drum had

become ulcerated and destroyed. This permitted the stratified squamous epithelium of the outer surface of the drum to progress around the edge of the perforation and to cover part of the ulcerated surface on the tympanic side of the drum.

In the air cell system of the middle ear extensive edema and infiltration of the mucosa by round cells and giant cells were present. Tubercles were noted at frequent intervals. Large areas of the mucosa were ulcerated. Areas of caseation were scattered about. The air space of the tympanum was almost completely filled with a fibrinopurulent exudate. The stapes footplate and secondary tympanic membrane were intact. The ossicular chain was buried in a mass of tuberculous granulation tissue. Early necrosis of bony trabeculae was noted. The mastoid air cell system had been largely removed surgically. Remnants of the air cells

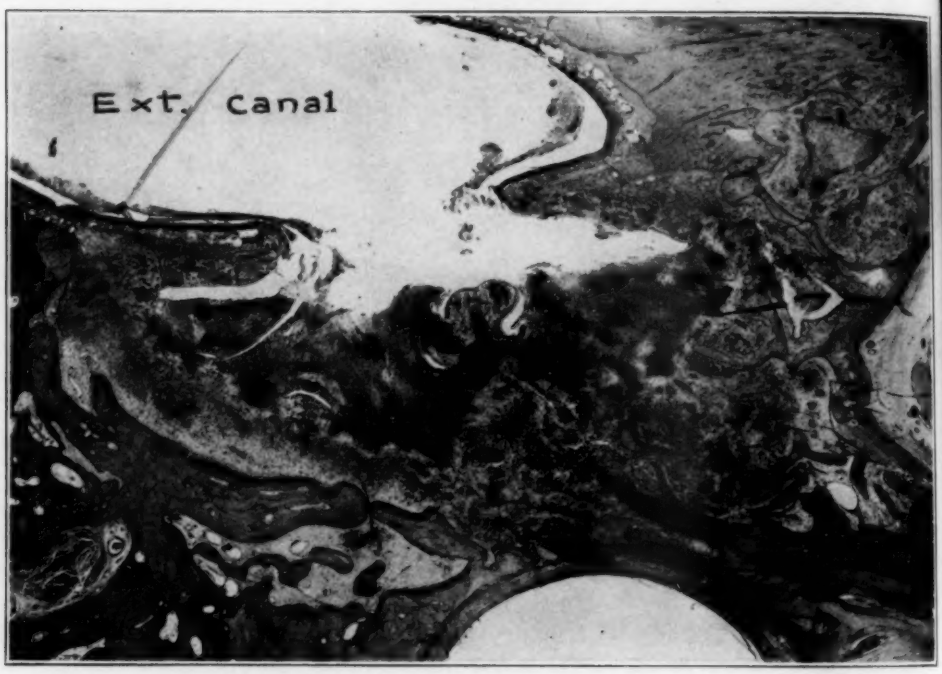

Fig. 6 (case 3).—Horizontal section (× 20) through the tympanum and perforated tympanic membrane of the right ear, showing extensive ulcerative tuberculosis. The mucosa is extensively ulcerated and has the character of a tuberculous granulation tissue covered by a thick layer of caseous exudate. Miliary tubercles are numerous. Stratified squamous epithelium has extended beyond the margins of the perforation and is invading the middle ear (fig. 7).

in the retrofacial region revealed marked edema of the mucosa, with scattered subepithelial fibrocaseous tubercles. Some of these tubercles had ruptured into the lumens of the air cells and were discharging tuberculous elements directly into the air spaces. The eustachian tube contained a fibrinopurulent exudate in its lumen. The tuberculous process was confined to the tympanic orifice of the tube; the region approaching the isthmus was free of involvement.

The petrosa was composed of young marrow and was not pneumatized. The marrow contained numerous conglomerate fibroplastic tubercles.

In the cochlea and vestibule a very early stage of serous labyrinthitis was evident.

In the internal auditory meatus a marked infiltration of the leptomeninges and nerve trunks was seen. Many giant cells were scattered throughout the nerve trunks, and a tendency to tubercle formation was noted.

(*b*) Left ear (figs. 8, 9 and 10): The pathologic changes throughout the left temporal bone were similar to those described in the right temporal bone.

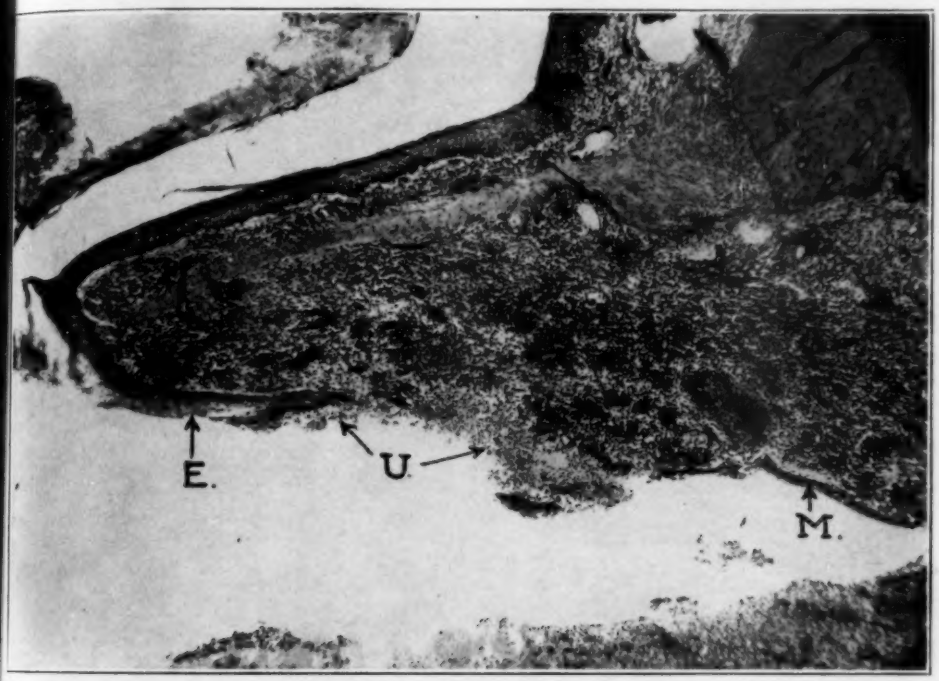

Fig. 7 (case 3).—Margin of a perforation in the right tympanic membrane (× 55) seen in figure 6. The epidermis from the external surface of the drum has grown over the margin of the perforation and into the middle ear, where it partly covers the adjacent ulcerated surface. Remnants of middle ear mucosa (*M*), ulcerated middle ear mucosa (*U*) and ingrowing epidermis (*E*) are shown.

The general observations in case 3 closely resemble those in case 2. The difference in size and age of the small metastatic tubercles was even more clearly indicative of repeated bacillemia. The demonstration of exudative and ulcerative features in the lungs, together with the proliferative meningeal reactions, speak for tuberculosis of some chronicity and for a childhood type of tuberculosis modified by the development of allergic immunity. The findings in the temporal bones support these conclusions.

In this case of advanced aural tuberculosis, the complexity of the final state does not permit "proof" of the successive steps in pathogenesis. The initial lesions were most probably hematogenous miliary tubercles located in the bone marrow (figs. 4 and 8) or mucous membranes or in both. Obviously, the enlargement of one or many of the mucosal tubercles must have led to ulceration and to the discharge of tubercle bacilli with exudate into the air spaces. The character of the subsequent spread within the complex system of air spaces is evident. Infection by bacilli in the exudate within the lumens was probably the

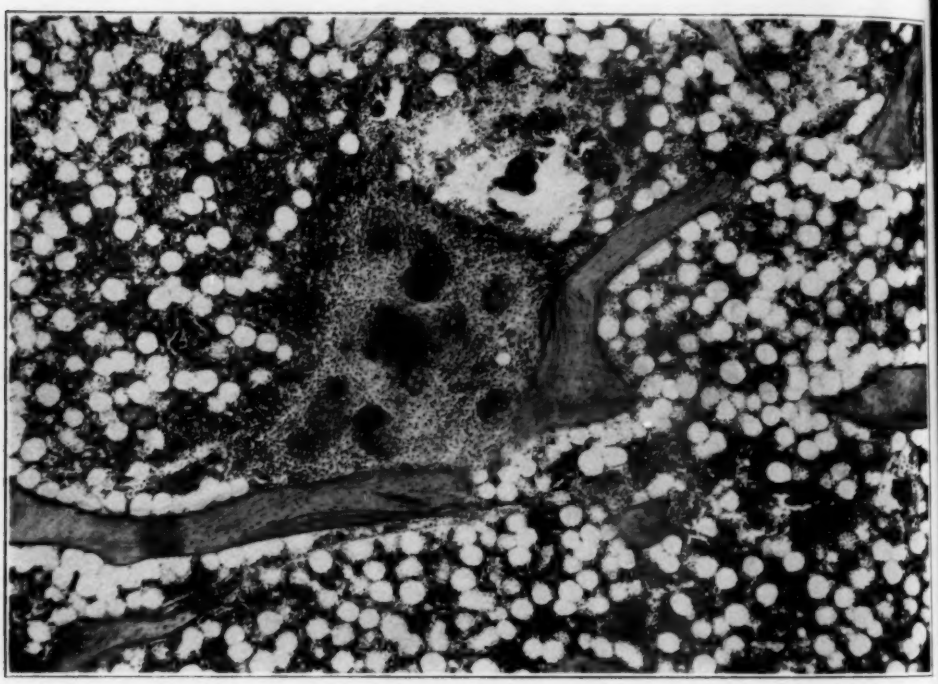

Fig. 8 (case 3).—Section (×60) showing a conglomerate small tubercle in the bone marrow of the petrosa in miliary tuberculosis. The tubercle contains epithelioid cells, numerous fibroblasts and areas of caseation. There is a slight absorption of the bony trabeculae in contact with the tubercle.

main mechanism—entirely analogous to the infection of the larynx or the intestine in ulcerative pulmonary tuberculosis. This does not exclude the participation of spread within the lymph channels of the membranes or of an occasional fresh hematogenous focus. Ultimately, almost all of the mucous linings become thickened and chronically inflamed, often with small tubercles scattered through them (figs. 5, 9 and 10). Many were focally or completely ulcerated, liberating more bacilli with

exudate (figs. 5 and 6). Some became plugged with caseous exudate which was organized to form tubercles (fig. 5). The most profound changes were noted in the epitympanum and mastoid antrum, where the exudate pooled. Here masses of caseous exudate overlay granula-

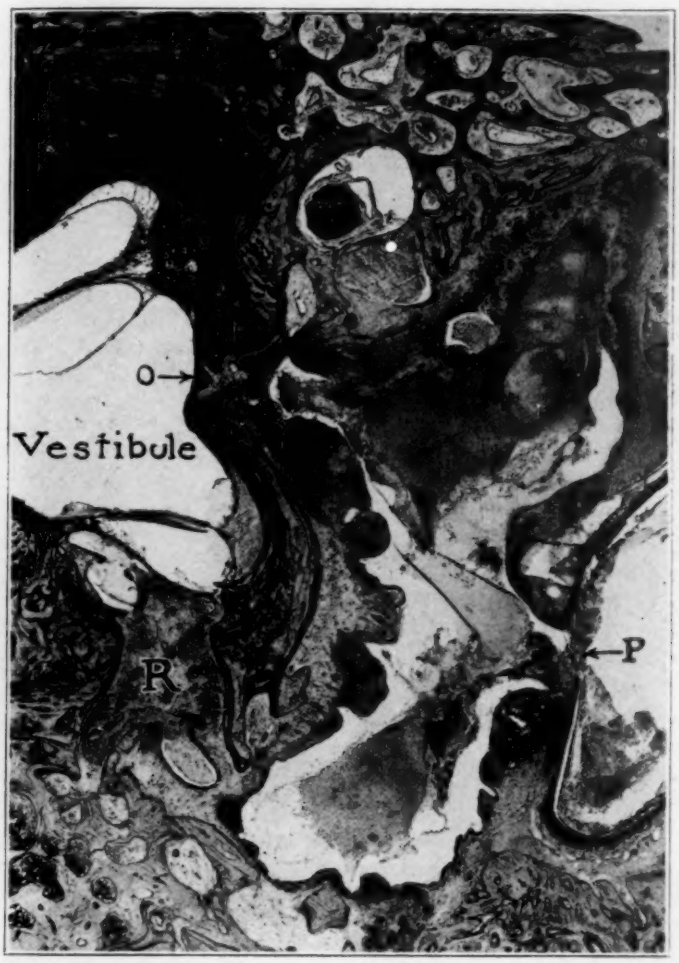

Fig. 9 (case 3).—Vertical section (× 55) from the left ear of a patient who had acute tuberculous otitis media in an advanced stage. Exudate fills the middle ear and adjoining air spaces. Tubercles and tuberculous ulcers are present in the greatly thickened mucosa. The niches of the round (*R*) and oval (*O*) windows are filled with tuberculous granulation tissue. There is a perforation (*P*) in the tympanic membrane.

tion tissue that thickened the membranes and obliterated portions of the cavity, entrapping the ossicles. The importance of the organization going on in this region should be stressed. Were such a process to go

on to healing, the outcome would be chronic adhesive middle ear disease. One other observation, too, needs to be emphasized. Squamous epithelium had grown from the external canal, through the perforation in the tympanic membrane, to extend for a considerable distance over the adjacent ulcerated tuberculous granulation tissue (figs. 6 and 7). The significance of such an observation with reference to possible subsequent development of a cholesteatoma, had healing occurred, is obvious.

The tuberculous changes in the eustachian tube were most marked at the tympanic orifice. Pathologic changes were noted to diminsh as

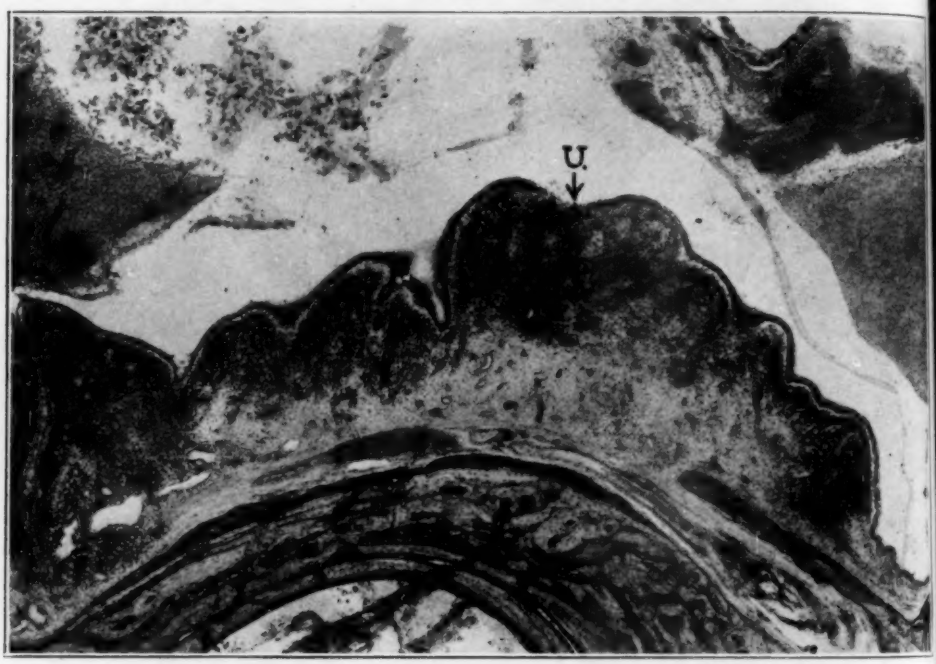

Fig. 10 (case 3).—Section (× 30) from the left ear. There is focal tuberculous ulceration of the mucosa over the promontory. The mucosa is thickened and polypoid in character. It is densely infiltrated by large mononuclear cells and lymphocytes. At the apex of the most prominent projection there is a freshly ulcerated caseous miliary tubercle (*U*) with epithelioid cells at its periphery. The exudate contains many large mononuclear cells and is focally caseous.

one approached the isthmus of the eustachian tube. This fact speaks against the theory that the disease was of tubal origin in this case.

Case 4.—N. M. was born a normal child at the Chicago Lying-in Hospital of the University of Chicago, on Nov. 14, 1937. He was returned to the department of pediatrics on March 15, 1938 with the complaint that five weeks previously a fever developed, which lasted only two days. Shortly thereafter a persistent cough and wheezing developed, which were present on the infant's admission to

the hospital. Roentgenograms of the chest revealed enlarged lymph nodes in both hili. The Mantoux test was positive. A diagnosis of pulmonary tuberculosis was made. The mother had a cough of two years' duration. Roentgenograms of her chest revealed three cavities. Her sputum was loaded with tubercle bacilli.

The infant improved steadily for a time but late in April began having a constant low grade fever. In the latter part of May a perineal abscess developed, which was incised and drained. Inoculation of guinea pigs with the pus obtained was later reported to have produced infection with the tubercle bacillus. After many attempts tubercle bacilli were found in gastric washings (May 24).

On August 16 otitis media developed on the left side. Myringotomy was done. Smears and culture of the pus obtained did not reveal the tubercle bacillus. On August 29 myringotomy was performed for otitis media on the right side. Both ears continued to discharge. Polypoid granulations developed in the left ear. Biopsy was done on two occasions but failed to show evidence of a tuberculous process.

In view of the persistent and profuse otorrhea, the formation of aural polyps, the daily fever, the steady loss of weight and the roentgen evidence of cellular breakdown in the left mastoid it was decided on October 18 to perform a mastoidectomy on the left side. The right ear had ceased to discharge. The cortex of the left mastoid bone was found to be intact. The trabeculae of the mastoid cells had been destroyed completely, and the cavity was filled with granulation tissue and pus. Histologic examination of the granulation tissue removed from the cavity revealed tuberculosis. Postoperatively the child improved for a short time. On November 3 signs of meningitis developed, which were confirmed by examination of the spinal fluid. On November 4 the left mastoid process was reopened. Granulations were removed. The dura was exposed and was found to be normal. An ossiculectomy was performed. Biopsy of the granulations again showed tuberculosis.

The patient did not improve postoperatively. The tuberculous meningitis progressed gradually until the patient died, on November 26. Permission for autopsy was refused.

The primary lesion of tuberculosis was probably in the lung, the infant having contracted the disease from his tuberculous mother. The perineal abscess, the otitis media and the meningitis were all tuberculous. The pulmonary symptoms appeared first, strongly suggesting a primary complex in the lung. The clinical course indicates that the primary lesion failed to resolve and resulted in hematogenous dissemination of the tubercle bacilli, which produced tuberculous otitis media, meningitis and perineal tuberculous abscess. The indications for surgical intervention in the cell system of the middle ear were open to question in this case. An attempt at eradication of a secondary focus, although apparently a very active one, is usually unsound unless the primary focus is definitely inactive. This is obvious when one realizes that tuberculosis of the temporal bone rarely, in itself, produces intracranial complications.

Case 5.—R. C., a youth aged 14, was first seen at the University of Chicago Clinics on Dec. 3, 1931. At the age of 6 years he was found to have tuberculosis of the ninth and tenth thoracic vertebrae. A year later Hibbs's fusion operation was performed. At the age of 12 years he underwent subtrochanteric osteotomy

for a tuberculous hip. At the time of the first admission to the University of Chicago Clinics arthrodesis was performed. At this time the results of otologic examination were negative.

When the youth was again seen, on Feb. 4, 1932, he was found to have otorrhea on the right side, with a perforation in the posteroinferior quadrant of the tympanic membrane. He was readmitted to the hospital on May 8 for an operation for epiphysial arrest. At this time his right ear was dry. After an uneventful postoperative course he was discharged.

His final admission was on July 23, 1932, when he complained of headache, vomiting and epigastric distress. Otoscopic examination revealed a dry central perforation of the tympanic membrane on the right side. There was no choking

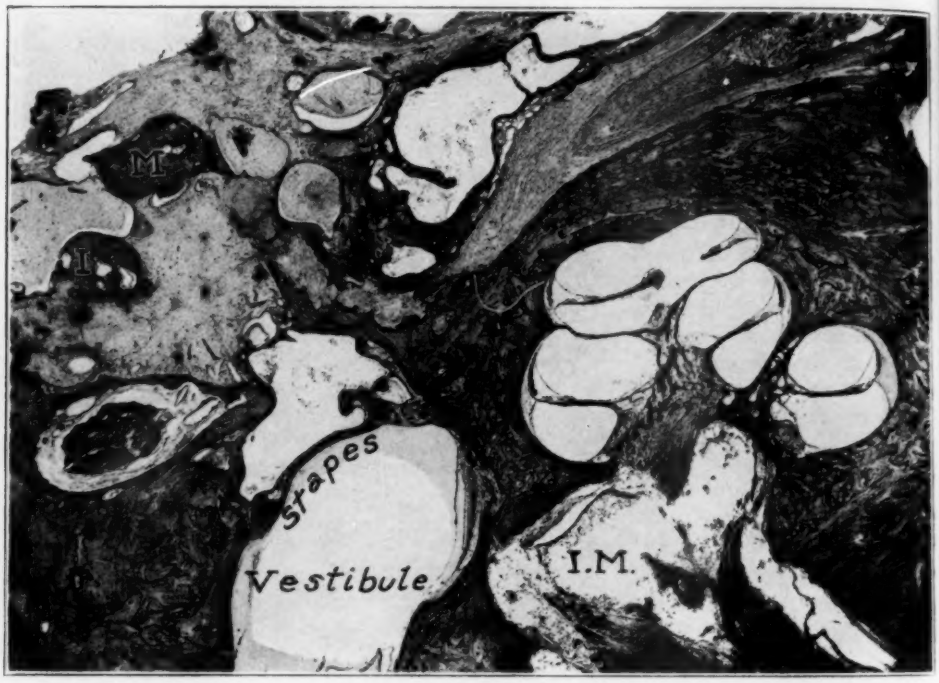

Fig. 11 (case 5).—Horizontal section (× 13) from the right ear of a patient who had tuberculous chronic adhesive otitis media. The footplate of the stapes, midmodiolar region of the cochlea, tensor tympani muscle and tendon, internal meatus (*I.M.*), vestibule, incus (*I*) and malleus (*M*) are shown. The tympanum about the ossicles is largely obliterated by mature fibrous tissue which contains sparse miliary tubercles and lymphocytes. Acute tuberculous meningitis is noted in the internal auditory meatus.

of the disk and no stiffness of the neck. Two days later examination of the spinal fluid revealed increased pressure (290 mm. of water), a cell count of 300 (chiefly lymphocytes) and a sugar content of 18 mg. per hundred cubic centimeters. A diagnosis of tuberculous meningitis was made, and Bokay (roentgen) treatment was instituted, without benefit. Seven days after admission that patient became comatose and on the thirteenth day he died.

Autopsy.—The postmortem observations were: an encapsulated fibrocaseous and partly calcified tubercle in the right lung (Ghon tubercle), with chronic fibroplastic (tuberculous?) inflammation of a right hilar lymph node; mild chronic fibroplastic tuberculosis in the apexes of both lungs, with fibrous adhesions about the right apex; sparse miliary fibroid and epithelioid tubercles in the tracheobronchial lymph nodes; small fibroid nodules, presumably healed miliary tubercles, in the spleen and liver; an old deformity of the lower part of the dorsal spine, without active tuberculosis; collapse and fibrous repair of vertebral bodies; operative fusion of the spinous processes; chronic fibroplastic and fibrocaseous tuberculosis of the left hip, with fibrous ankylosis; subacute tuberculous leptomeningitis and meningoencephalitis, and chronic tuberculous adhesive otitis media and mastoiditis.

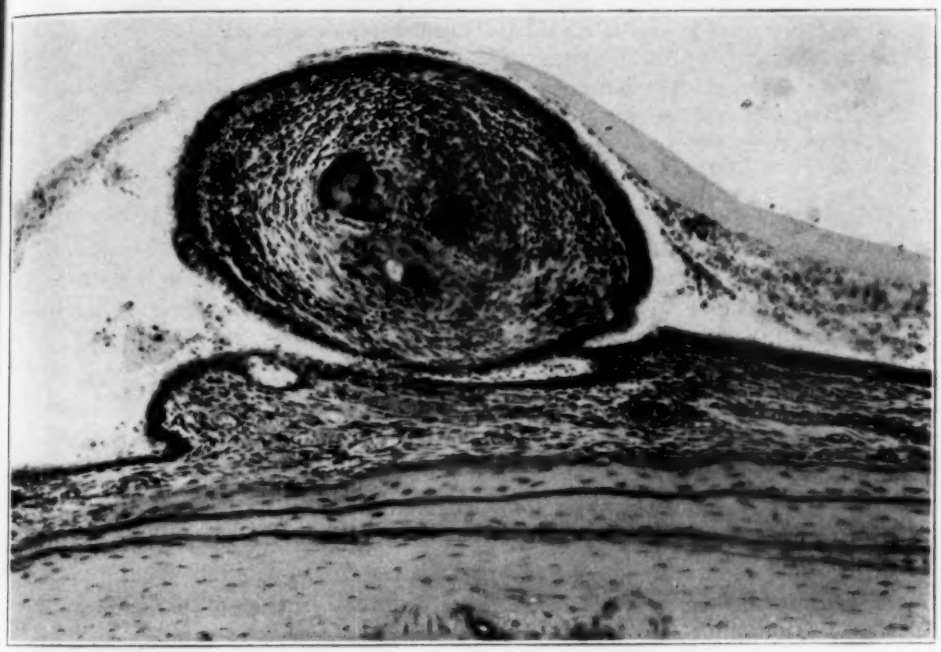

Fig. 12 (case 5).—Section (× 150) from the right ear, showing a miliary tubercle in the mucosa of the posterior wall of the tympanum. The adjacent mucosa shows mild chronic inflammation with very little thickening. The core of the polyp contains a tubercle composed of epithelioid cells and Langhans giant cells, with a mantle of fibroblasts and lymphocytes.

Microscopic Examination.—(*a*) Right ear (figs. 11 and 12): In the tympanic membrane was a large perforation. Remnants of the drum membrane showed tuberculous changes, with round cell and giant cell infiltration. Pathologic changes of the air cell system of the middle ear were most marked in the epitympanum and in the region of the mastoid antrum, where there was found considerable fibrous granulation tissue containing many small round cells and giant cells. There was no evidence of caseation, although the disease was still active. Free pus was present in the air spaces, but it was very thick and appeared to have been

retained for a long period. Fibrous adhesions had formed in various portions of the epitympanum. There was evidence both of bone resorption and of new bone formation. Many of the bony trabeculae were thicker than normal. The ossicular chain was buried in fibrous granulation tissue. The footplate of the stapes and the secondary tympanic membranes were intact. There was no necrosis of the labyrinthine capsule, nor was there evidence of extension into or from the intracranial cavity. The mucosa of the eustachian tube was not involved. In the mastoid process pathologic changes were more marked in the region about the antrum, which was characterized by small round cell and giant cell infiltration and considerable fibrosis.

The petrosa was composed of fatty marrow. No foci of miliary tuberculosis were found.

On examination of the internal ear a mild serous exudate containing a few lymphocytes was present in the basal coil of the cochlea, adjacent to the cribriform plate.

(*b*) Left ear: The air cell system of the middle ear was normal. In the cochlear and vestibule serous labyrinthitis in a very early stage was noted. The petrosa was composed of fatty marrow, with no evident miliary foci. The condition of the internal ear was the same as that noted in the right temporal bone.

It is likely that in this case the initial infection was not extensive. It was represented at autopsy by a partially healed Ghon tubercle and a small fibroid hilar component constituting a primary complex of limited extent. Despite this relative benignity tuberculous bacillemia must have occurred, to account for the multiple metastatic foci. These metastatic lesions were remote, in tissues isolated from natural portals of entry of the tubercle bacilli, and were not located in the course of lymphatic drainage from the Ghon tubercle. The episodes of the clinical history reflect the fate of the osseous metastases. At the time of autopsy the spinal lesion was clinically and anatomically healed, while mild proliferative tuberculosis persisted in the hip. The remaining tuberculous foci must have resulted from hematogenous spread from either the primary or the metastatic osseous lesions. These included sparse healed tubercles in the liver and spleen, the asymptomatic pulmonary apical tuberculosis, which was of a mild chronic adult or reinfection type, and the aural lesion, which had ceased to cause symptoms three months before the patient's death. The exact pathogenesis of the meningitis was not ascertained. Subacute tuberculous leptomeningitis, occurring in the absence of miliary tuberculosis (for which there was no evidence), is usually due to one of two processes. There may be transdural extension from a contiguous osseous lesion or there may be spread of bacilli to the subarachnoid space from a small cerebral tuberculoma. In this case there was no evidence of spread from osseous structures, including the temporal bone. There were regions where both the meninges and the subjacent brain were involved by tuberculosis. It was impossible to ascertain which involvement was the older.

In this case the marrow of the petrous apexes was of the fatty type, which is less likely to become the site of miliary foci because of the scanty blood supply. This fact plus the sparse miliary dissemination probably accounts for the absence of miliary foci in the marrow spaces of the petrous apexes.

The importance of this case is that it demonstrates that the tubercle bacillus may cause chronic adhesive otitis media. Moreover, despite the persistence of infection, as shown by the presence of active tubercles, there was a remarkable tendency to healing. It is evident that seemingly nonspecific chronic adhesive otitis media in tuberculous patients may be of tuberculous origin. It is even possible and not unlikely that the same is true occasionally of adhesive otitis in patients who are not known to have tuberculosis. This statement is made in view of the prevalence of subclinical, mild tuberculosis of the primary or of the reinfection type and in view of the evidence that transitory mild bacillemia is a common accompaniment of tuberculous infection.

Case 6.—E. H., aged 6 years, was first seen in the University of Chicago Clinics on Feb. 26, 1932, complaining of a painful lump on the back, which had been present for six months. A diagnosis of Pott's disease was made, and the patient was admitted to the hospital. Otologic examination at this time revealed chronic suppurative otitis media and mastoiditis on the left side. Four days later a body cast was applied. The patient was discharged to a convalescent hospital. He was readmitted three and one-half months later with a fever and headache of a week's duration. At the time of admission the patient was unconscious. A diagnosis of tuberculous meningitis was made. Roentgen studies of the chest failed to reveal evidence of miliary tuberculosis. The patient died fourteen days after admission.

Autopsy.—The observations at autopsy were a small calcified tubercle in the left lung (Ghon tubercle); encapsulated fibrocaseous tubercles in the right lung and in the right hilar lymph nodes; acute and chronic hematogenous miliary tuberculosis; subacute tuberculous leptomeningitis; disseminated epithelioid and fibroplastic miliary tubercles in the liver, spleen, bone marrow, lungs, leptomeninges and kidneys; chronic destructive tuberculosis of the dorsal spine; extradural caseous abscess in the spinal canal; focally calcified encapsulated paravertebral abscess, and asymmetry of the skull, with flattening of the squamous portion of the left temporal bone.

Microscopic Examination.—(*a*) Left ear (fig. 13): The tympanic membrane had been largely destroyed, both by disease and by injury sustained in the removal of the specimen at autopsy.

The middle ear was lined by stratified squamous epithelium, which was continuous with the epidermis of the external auditory canal. This epithelium covered the ossicles and extended into the attic, where collections of exfoliated epithelium were present. There was no histologic evidence of tuberculosis in the middle ear or mastoid air cells. The mastoid cells adjacent to the tympanum showed edema and fibrous thickening of the mucosa.

The internal ear revealed a few plasma cells in the basal coil of the cochlea, adjacent to the cribriform plate.

In the marrow spaces of the petrosa were several foci of fibroplastic and fibrocaseous miliary tubercles.

On examination of the internal auditory meatus the leptomeninges were seen to be infiltrated by lymphocytes and large mononuclear cells.

(*b*) Right ear: The tympanic membrane was normal.

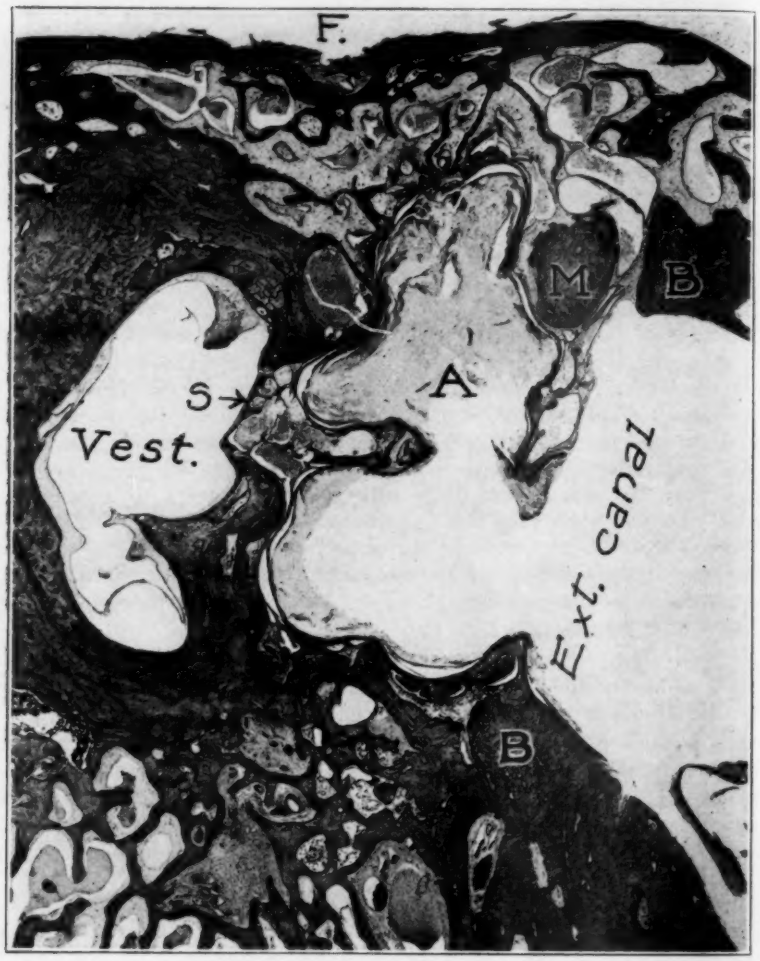

Fig. 13 (case 6).—Vertical section (× 13) from the left ear of a child who had a cholesteatoma, with Pott's disease. The middle ear (*A*) and attic are lined by epidermis which has extended in through the perforation from the external canal. Considerable desquamated epithelium has collected in the attic. There is no evidence of tuberculosis in the middle ear cell system, but tubercles are present in the marrow. The annulus tympanicus (*B*), malleus (*M*), stapes footplate (*S*) and middle fossa (*F*) are shown.

The air cell system of the middle ear revealed mild fibrous thickening and infiltration of the mucosa in the cells of the epitympanum. A small amount of purulent exudate was present in the tympanic cavity.

The mastoid air cells were normal. There was no evidence of tuberculosis in the air cell system.

On examination of the internal ear a small amount of serous exudate and a few plasma cells were present in the basal coil of the cochlea, near the cribriform plate.

In the marrow spaces of the petrosa fibrocaseous and fibroplastic miliary tubercles were noted.

The condition of the internal auditory meatus was the same as that described for the left temporal bone.

Tuberculous infection must have been present in this patient long before symptoms were noted. Chronicity is evidenced by the calcifications in the Ghon tubercle and in the paravertebral abscess and by the encapsulated fibrocaseous tubercles in the right lung and hilar lymph nodes. There is clinical evidence of chronicity in that there were long intervals between the episodes of hematogenous dissemination. Many of the internal organs contained old fibroid tubercles as well as young ones. The terminal dissemination was most probably bacteria derived from the caseous spinal lesion. Although the disease process had broken through into the spinal canal and lay in contact with the dura, the microscopic evidence favored the conclusion that the leptomeninges were infected by the blood stream route. The flattening of the squama of the left temporal bone is evidence that the left aural lesion was of considerable chronicity.

The single and conglomerate miliary tubercles found in the petrous apexes were similar to those found elsewhere. It is reasonable to assume that they resulted from successive episodes of hematogenous dissemination and formed but part of the picture of generalized miliary tuberculosis. The right middle ear showed only early otitis, with no specific features. The tympanic membrane of the left ear had been destroyed, and the middle ear showed the chronic changes associated with cholesteatoma. The conditions observed in case 5 demonstrated that chronic adhesive otitis may result from tuberculous infection of the middle ear. In case 3 it was shown that the first phase of cholesteatoma formation may develop while tuberculous otitis is still active and progressing. Case 6 is presented not to give a proved example of cholesteatoma caused by tuberculosis but to emphasize the possibility that such a lesion may have a tuberculous origin. It should be noted that there are reports of cholesteatoma developing in association with tuberculosis of the middle ear.[14] The high incidence of chronic suppuration of the middle ear in

14. Bárány, R., cited by Retrouvey, H.: La tuberculose de l'oreille, Rev. de laryng. **48**:481, 1927. Oppikofer, E.: Fünfte Jahresversammlung der Gesellschaft deutscher Hals-, Nasen- und Ohrenärzte, Munich, 1925; cited by Brüggemann.[9e] Ulrich, cited by Nager, F. R.: The Cholesteatoma of the Middle Ear: Its Etiology, Pathogenesis, Diagnosis and Therapy, Ann. Otol., Rhin. & Laryng. **34**:1249, 1925.

tuberculous persons (4.7 to 11 per cent) suggests that in many of these the aural condition may have been tuberculous in character at the onset.

CASE 7.—I. T., an 11 year old Mexican girl, came to the University of Chicago Clinics on Dec. 13, 1938. Three years previously a cold abscess of the scalp appeared immediately above the left auricle. This gradually extended about the auricle until it involved the preauricular, postauricular and upper cervical regions on the left. On several occasions the abscess was incised. Early in the course of the disease a piece of bone sloughed out through one of the draining sinuses. Multiple sinuses developed. Roentgenograms of the chest, guinea pig inoculation of the discharge from the sinuses and the Mantoux test all failed to reveal tuberculosis. Otherwise, the child was in good health.

Otologic examination when the child was first seen by us revealed eight draining sinuses about the left ear. For at least two years she had had a fetid otorrhea from the left external auditory canal and the hearing in the left ear had been depressed. No hearing in the left ear by either air or bone conduction was recorded on the audiometer when the right ear was masked with the Bárány apparatus. The left external auditory canal was partly collapsed. Its lumen was filled by a large aural polyp. Biopsy of the polyp later revealed tuberculosis. Guinea pig inoculation of tissue from one of the sinus tracts later gave a positive result. Roentgen studies of the temporal bones showed an extensive middle ear cell system on the normal side, invading the perilabyrinthine areas and the apex. The diseased ear showed a similar extent of pneumatization, with a marked increase in density, throughout the temporal bone. Roentgenograms of the chest demonstrated nothing abnormal except a possible tuberculous lesion in the first thoracic rib. The tuberculin test was positive. The child was sent to a convalescent children's home for six months. The fistulas failed to heal during this interval.

She was readmitted to the University of Chicago Clinics on June 16, 1939. Three days later radical mastoidectomy was performed on the left. Three cortical fistulas were found. The mastoid cavity was filled with irregular tracts of granulation tissue, running between masses of very hard bone. The lateral sinus plate was found to be eroded, exposing the lateral sinus. The middle fossa plate was eroded from the tegmen antri to the roof of the canal of the tensor tympani muscle. The mastoid cells were completely exenterated and a radical operation performed. The attic and middle ear were filled with fibrous granulation tissue. The peritubal cells were extensive and were removed, exposing the ascending portion and knee of the carotid artery. The apex was not entered.

The postoperative course was uneventful, and in three months all of the fistulas had healed. At the time of writing there has been no subsequent discharge from the healed fistulas. The middle ear cavity has been dry for nearly two years.

The case is illustrative of tuberculosis of the temporal bone which healed after surgical intervention. The child was at rest in bed and was maintained on a high caloric and high vitamin diet, for six months before operation, without any appreciable improvement in the local lesion. The aural infection may have been on a hematogenous basis. However, in this case it is not possible to determine the exact origin of the aural lesion. The sputum was not infected, and examination of the entire family for tuberculosis gave negatiye results. The lesion in the thoracic rib was suggestive of tuberculosis, and if tuberculosis was

present it unquestionably was of hematogenous origin. Our prolonged observation (i. e., for six months) failed to reveal any tuberculous activity of significance elsewhere in the body. This fact plus the fact that the local lesion in the left temporal bone did not resolve influenced us in performing radical mastoidectomy to eliminate the foci of disease. The operative findings illustrate how extensive the destruction of the temporal bone may be without producing an intracranial complication.

Case 8.—L. D., a 5 year old boy, first came to the University of Chicago Clinics on March 12, 1932. Eight weeks previously a swelling of the right cervical glands developed. This ruptured spontaneously in three weeks. The glands drained for four days and then resolved. Four weeks previous to admission the left ear began discharging spontaneously without concomitant pain or fever. Two weeks later the left cervical and preauricular lymph nodes began to enlarge. Four days previous to admission these enlarged lymph nodes were incised and pus was obtained.

Examination at the time of admission revealed the draining abscesses in the preauricular and cervical regions, profuse otorrhea in the left ear, edema of the walls of the external auditory canal on the left side and induration, inflammation and tenderness over the left mastoid. Roentgenograms of the left mastoid showed complete clouding of the mastoid and petrosa.

On March 15 mastoidectomy was performed. The cortex was intact. A large abscess was found extending from the antrum to the mastoid tip. There was a small erosion of the lateral sinus plate. At this time no communication was established between the preauricular sinus and the region of the zygomatic cells.

Postoperatively the child had a daily rise of temperature to 39 C. (102.2 F.). The cervical, preauricular and mastoid wounds continued to discharge. On March 25 the preauricular sinus was found to communicate with the mastoid wound. Repeated tuberculin tests were negative. On several occasions culture of discharge from the left ear revealed Streptococcus haemolyticus.

On April 20 the mastoidectomy cavity was reopened. It contained some thick caseous pus. Granulation tissue and necrotic bone over the lateral sinus were removed. The preauricular sinus was curetted. Smears, cultures and guinea pig inoculation subsequently failed to show the tubercle bacillus. Microscopic examination of the granulation tissue removed at operation revealed tuberculosis. Roentgenograms of the chest disclosed no evidence of tuberculosis in the periphery of either lung, but did show evidence of tuberculous involvement of the hilar lymph nodes on the right (primary complex).

Postoperatively the child improved gradually until on November 29 the several sinuses were healed completely. There was no recurrence until March 6, 1939, when the patient returned with fluctant postaural swelling on the left. This subsided spontaneously in a few days and probably represented nonspecific recurrent mastoiditis.

In this case presence of tuberculosis was not suspected at the time of admission. The course of the disease was characteristic of some forms of tuberculosis of the temporal bone. The slow onset, cervical adenitis, preauricular adenitis and insidious onset of otorrhea without pain or fever are all typical of this variety of aural tuberculosis. The roentgenograms of the chest strongly suggest a primary complex in the lung. At

the time of writing the impression is that the pulmonary focus was responsible for a hematogenous dissemination which involved the left ear.

PATHOGENESIS

The initial lesion in tuberculosis of the temporal bone occurs as a single focus, or more often as multiple foci, of miliary tuberculosis, in the marrow spaces, in the mucosa of the air cell spaces of the middle ear or in both. It is possible that infection in the bone marrow may spread, by contiguity or by way of lymphatics, along vascular channels to the mucosa of an air cell. At this point a subepithelial tubercle develops. This tubercle in the mucosa (hematogenous or secondary to a lesion in the marrow) may result in lmyphogenous spread of tubercle bacilli beneath the mucosa, or the initial mucosal tubercle may ulcerate and discharge infected material into the air cell spaces. Once the infected exudate enters the air cell system, it spreads and may become implanted into the mucosa at multiple sites (this process is analogous to the infection of the larynx by infected sputum in patients with pulmonary tuberculosis). Eventually the disease process concentrates in the regions of the mastoid antrum and epitympanum. This localization may be due to (1) the lymphatic drainage from the mastoid air cell system to the antrum and epitympanum, (2) the presence of ossicles with their accompanying ligaments and folds of mucous membrane, which favor the collection of exudate, (3) the movements of the ossicular chain or (4) a richer vascular supply at this point.

It is conceivable that in many cases the tubercles in the bone marrow may remain localized and eventually heal, resulting in a clinical cure. In a number of cases which we have not reported here, we have found old healed miliary tubercles in the marrow spaces of the temporal bone.

Tuberculosis of the air cell system of the middle ear may result in (1) chronic tuberculous otitis, (2) replacement by a chronic nontuberculous infection, (3) development of a chronic adhesive middle ear disease or (4) development of a cholesteatoma.

COMPLICATIONS

Complications resulting from tuberculosis of the temporal bone are unusual. They are a sign of progression of the disease. The following types of complication have been reported: single or multiple fistulas about the auricle, due to extension from the cell system and regional lymph glands; facial paresis or paralysis due to chronic interstitial and parenchymatous neuritis;[13c] labyrinthitis due to direct erosion of the capsule or the windows[15]; circumscribed tuberculous pachymeningitis;

15. Zange, J.: Pathologische Anatomie der mittelohrentspringenden Labyrinthitis, Wiesbaden, J. F. Bergmann, 1919.

erosion of the internal carotid artery or of the jugular bulb; tuberculous osteomyelitis of the petrous pyramid due either to confluence of multiple miliary tubercles or to direct extension from the middle ear spaces [16] and extension to the temporomandibular joint.

The dura is extremely resistant to intracranial extension of tuberculosis from the temporal bone. We have failed to find in the literature a case in which it was proved histologically that diffuse tuberculous meningitis was due to direct extension from the temporal bone.

DIAGNOSIS

In the early stages of tuberculosis of the ear diagnosis may be difficult. The presence of pulmonary tuberculosis, tuberculous meningitis or some other tuberculous condition may first arouse suspicion of a specific infection in the ear. There is a wide variation in the onset and course of the disease.

Blegvad [17] described a characteristic appearance of the tympanic membrane in tuberculous otitis media which permits a definite diagnosis before the appearance of otorrhea and granulation tissue. More recently Lüscher and Rotmann [18] described characteristic otomicroscopic findings in tuberculous otitis media. Some authors [19] have reported roentgen changes which are typical of tuberculosis of the ear. However, Worning and Jörgensen [20] were unable to diagnose tuberculosis of the temporal bone solely on roentgen evidence. Our histologic studies show that roentgen studies could not be expected to offer reliable evidence in differentiating tuberculosis from nonspecific infection of the cell system of the middle ear.

Bacteriologic and histologic examination offer the best means of establishing a diagnosis. Direct smears, cultures and guinea pig inoculation may demonstrate the presence of the organism. Biopsy of granulation tissue may establish the diagnosis. In some cases, par-

16. Lombard, cited by Retrouvey, H.: La tuberculose de l'oreille, Rev. de laryng. **48**:481, 1927. Hayden, D. B., and Cohen, A. I.: Tuberculosis of the Middle Ear with Report of a Case, Laryngoscope **35**:883, 1925.

17. Blegvad, N. R.: Tuberculosis of the Middle Ear, Acta oto-laryng. **24**:187, 1936; Le diagnostic précoce de la tuberculose de l'oreille moyenne, Ann. d'oto-laryng., 1939, p. 804.

18. Lüscher, E., and Rotmann, S. S.: Typische otomikroskopische Befunde der Mittelohrtuberkulose, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:10, 1935.

19. Mayer, E. G.: Otologische Roentgendiagnostik, in Holzknecht, G.: Handbuch der theoretischen und klinischen (allgemeinen und speziellen) Röntgenkunde, Berlin, Julius Springer, 1930, vol. 2. Thienpont, R.: Au sujet du radiodiagnostic de la tuberculose de l'oreille, Ann. d'oto-laryng., 1939, p. 808.

20. Worning, B., and Jörgensen, A.: Der Wert der Röntgenuntersuchungen bei entzündlichen Erkrankungen im Os temporale, Ztschr. f. Laryng., Rhin., Otol. **260**: 325, 1935.

ticularly those in which the disease is subsiding or has healed, the diagnosis can be established only by histologic examination of the temporal bone.

TREATMENT

The overwhelming evidence that aural tuberculosis is secondary to a primary focus elsewhere provides the guide to treatment. Attention should be directed primarily toward improving the general condition of the patient. Tuberculosis in the temporal bone rarely threatens life, it frequently heals spontaneously and there is little evidence that it has an adverse effect on the course of the primary source of infection. If the general condition is good and if the lesion in the temporal bone is the chief active focus in the body, then adequate surgical treatment may be indicated. If poor surgical judgment is used, there is danger that an exacerbation of the local or of a distant focus may be produced inadvertently by the surgical procedure. Surgical intervention, as well as superimposed infectious processes (such as measles and scarlet fever), may precipitate a miliary dissemination.

The extent of the surgical procedure is dependent on the extent of the disease. An attempt should be made to remove the tuberculous focus as completely as possible. If the cell system of the middle ear is involved, a thorough exenteration is required.

SUMMARY

According to statistics, which obviously err on the side of conservatism, in less than 3 per cent of all cases of otitis media has this condition been due to tuberculosis and only 1 to 3 per cent of patients with pulmonary tuberculosis have had otitis media that was diagnosed as tuberculous.

On the other hand, the incidence of supposedly nonspecific suppurative otitis media in patients with pulmonary tuberculosis is unusually high (4.7 to 11 per cent). It is highly probable that in many of these cases the otitis media was originally tuberculous.

A series of cases are presented which illustrate the pathogenesis and character of tuberculosis of the temporal bone.

Tuberculosis of the temporal bone usually originates by the hematogenous route. As with other hematogenous lesions, the source of tubercle bacilli is usually a caseous tuberculous focus. Such foci are most often the caseous lesions of a primary infection in the lung and hilar lymph nodes or else of a primary infection in the mesenteric or cervical lymph nodes. However, any focus, and above all any active caseous focus (primary or secondary), may be the source from which tubercle bacilli enter the blood stream.

Bone marrow, including that of the temporal bones, shares with the liver and spleen the major histiocytic activity of the reticuloendothelial system. Hence, in a sense these tissues act like filter beds with respect to bacteria circulating in the blood stream. As a corollary of this function, in miliary tuberculosis these organs are especially common sites of miliary tubercles.

The initial lesion of tuberculosis of the temporal bone occurs as a single focus or more often as multiple foci of miliary tuberculosis, in the marrow spaces of the temporal bone, in the mucous membrane of the air spaces of the middle ear or in both.

Routine histologic examination of the temporal bone in persons with miliary tuberculosis will reveal a very high incidence of foci of miliary tuberculosis in this structure.

Histologic evidence is offered to support our contention that healing of tuberculous otitis media may in some cases result in either the formation of cholesteatoma or the development of a chronic adhesive middle ear disease.

Dr. Eleanor Humphreys, of the department of pathology of the University of Chicago, aided in the interpretation and discussion of the general pathologic aspects of the cases presented in this paper.

ACUTE SURGICAL MASTOIDITIS

REVIEW OF CASES, WITH SPECIAL REFERENCE TO POSTOPERATIVE HEARING AND TO THE ROLE OF SULFANILAMIDE AND ITS DERIVATIVES IN THERAPY

ROBERT E. PRIEST, M.D.
MINNEAPOLIS

A study of acute surgical mastoiditis at the University of Minnesota Hospitals is presented here. It was based on two groups of case records. The first of these was analyzed for data pertaining to incidence and covers the period from 1929 to 1939. The second group comprised the records of 298 patients treated during a four year period, from 1936 to 1940. A detailed analysis of the second group takes in several factors, including

TABLE 1.—*Annual Incidence of Acute Surgical Mastoiditis*

Year	Number of Cases
1929	48
1930	53
1931	27
1932	35
1933	49
1934	48
1935	85
1936	100
1937	70
1938	64
1939	69

especially postoperative hearing ability and the effect of sulfanilamide and its derivatives on therapy. A report by Boies [1] in 1936 covered the 240 cases immediately preceding this group.

The series presented here is not large. The percentile figures would probably not hold with complete accuracy if compared with those based on a larger group. It is thought that they do indicate trends. Hence, they are included.

INCIDENCE

The incidence of acute surgical mastoiditis at this hospital for a ten year period is seen in table 1.

Submitted to the Faculty of the Graduate School of the University of Minnesota in March 1941 in partial fulfilment of the requirements for the degree of Master of Science in Otolaryngology.

1. Boies, L. R.: Extradural Inflammation: A Study of Its Occurrence in Acute Surgical Mastoiditis, Tr. Am. Acad. Ophth. **41**:150-162, 1936.

The table shows a fairly constant annual number of cases until 1935-1936, when measles and scarlet fever caused a sharp increase. The incidence decreased in 1937 and remained fairly steady thereafter.

Spink [2] expressed the belief that sulfanilamide began to be used widely at University Hospitals about the middle of 1937. No appreciable effect of the use of this drug on the incidence of acute surgical mastoiditis at this hospital is now apparent. I do not know how widely the drug was used outside the larger centers in 1938 and 1939. Most of the patients treated at the University Hospitals come from small communities. The future may show a decrease in the number of cases of this disease due to more adequate use of sulfanilamide and its derivatives in cases of acute otitis media. Experience at the University of Pennsyl-

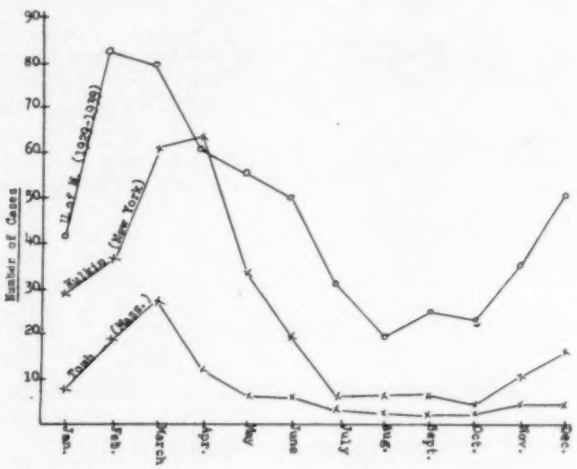

Chart 1.—Seasonal incidence of mastoiditis.

vania Hospital, Philadelphia, is exactly similar to that at the University Hospitals. Houser,[3] in 1939, said, "From these figures one concludes that preoperative administration [of sulfanilamide] has not appreciably changed the curve of incidence of acute surgical mastoiditis in this hospital." He went on to say that many of their patients are neglected before reaching the hospital, where advanced disease is often found on admission. Hence, his report is not necessarily a brief against the use of sulfanilamide in otitis media.

The seasonal incidence of acute surgical mastoiditis follows a rather definite pattern. Chart 1 shows the ten year incidence curve (1929-

2. Spink, W.: Personal communication to the author.

3. Houser, K. M.: A Survey of the Use of Sulfanilamide in Acute Otitis Media, Laryngoscope **49**:825-832 (Sept.) 1939.

1939) at the University Hospitals, together with curves from other localities.[4] The remarkable similarity in the shapes of the graphs is apparent. The number of cases is low during August, September and October. It rises during November, December and January, to reach its peak during February, March and April. Through the early summer it declines gradually to the August low point.

This general pattern of seasonal incidence agrees with that noted by other authors whose graphs are not shown on the chart.[5] A noteworthy exception is the chart given by Rawlins and Reich,[6] of San Francisco. Their graph of seasonal incidence has not only the usual single cold weather peak but also a second, equally high rise in July. The influence of the climatic factor on mastoiditis has been discussed by Fabricant.[7]

TABLE 2.—*Age Incidence of Acute Surgical Mastoiditis*

Age	Number of Cases
Less than 1 year	10
1 - 3	40
4 - 5	34
6 - 10	69
11 - 20	74
21 - 30	29
31 - 40	16
41 - 50	15
51 - 60	9
61 - 70	2
Over 70	2

Work to show that the characteristics of organisms may vary with their climatic environment has been reported recently by Lynch.[8] The age incidence appears in table 2.

4. (*a*) Kulkin, S.: Acute Mastoiditis from a Surgical Point of View, Arch. Otolaryng. **29**:306-313 (Feb.) 1939. (*b*) Tomb, E. H.: Simple Mastoidectomy: A Critical Study of One Hundred Consecutive Cases, Arch. Otolaryng. **31**:478-490 (March) 1940.

5. Kopetzky, S. J., and Hadjopoulos, L. G.: Relationship of Upper Respiratory and Alimentary Tract Flora to Mastoid Infections, wtih Particular Reference to the Epidemiology of Mastoiditis, Laryngoscope **42**:661-673 (Sept.) 1932.

6. Rawlins, A. G., and Reich, A.: Acute Surgical Mastoiditis with Résumé of Four Hundred and Three Operated Mastoids, Tr. Pacific Coast Oto-Ophth. Soc. **23**:85-107, 1938.

7. Fabricant, N. D.: Mastoiditis: Climatic Factor, Arch. Otolaryng. **30**:549-556 (Oct.) 1939.

8. Lynch, M. G.: Geographic Virulence of Mastoiditis, J. A. M. A. **115**:826-828 (Sept. 7) 1940.

The greatest incidence occurs in persons under 20 years of age. This agrees with other reports.[9]

TYPE OF OPERATION

Operations for acute surgical mastoiditis are done to relieve the immediate toxic symptoms, to eliminate the danger of intracranial and blood stream extension and to permit the ear to heal completely with as nearly normal hearing as possible. These purposes are best accomplished by the complete removal of cells down to the inner cortex of the temporal bone. When a mastoidectomy is done completely, dural exposure is common. In this series the incidence of dural exposure was as shown in table 3.

The figures just given agree closely with those published elsewhere.[10] They are important because in conjunction with the low mortality figures in the present series they prove that dural exposure can occur safely despite the contiguous mastoid infection.

TABLE 3.—*Incidence of Exposure of the Dura*

	Middle Cranial Fossa	Sigmoid Portion of Lateral Sinus
Surgical	105 (35%)	76 (25%)
By necrosis of bone	15 (5%)	74 (25%)
Total	120 (40%)	150 (50%)

One justification for doing complete mastoidectomy rather than simple drainage is found in the records of patients having recurrent mastoiditis. In the 17 cases (6.6 per cent) of recurrence in our series simple mastoidectomy had been performed previously. In every instance the first operation had been incompletely done. Three of the patients had been operated on at the University Hospitals and 14 elsewhere. Obviously, not every patient who is incompletely operated on will have a recurrence of mastoiditis. It is significant, however, that no recurrence was encountered in patients who had been submitted to a complete operation. In some of the cases postauricular abscess accompanied a recurrence of acute otitis media. In these, recovery followed simple incision and drainage. In every case in which a second mastoidectomy was required, large groups of cells were found which should have been removed at a complete first operation.

9. (*a*) Coates, G. M.; Ersner, M. S., and Persky, A. H.: A Study of Mastoiditis: A Résumé of Six Hundred and Forty-Four Cases, Laryngoscope **40**:245-257 (April) 1930. (*b*) Boies.[1] (*c*) Tomb.[4b] (*d*) Rawlins and Reich.[6]

10. Boies.[1] Tomb.[4b]

These findings are in agreement with those of Lillie [11] and of Lacy.[12] In 1928 Lillie [11] reported 53 cases of reinfection of the middle ear and mastoid encountered at the Mayo Clinic. In 50 of these 53 cases an incomplete mastoidectomy had previously been performed elsewhere.

Lacey [12] reported 50 cases of recurrent mastoiditis studied in the department of otolaryngology of the University of Kansas Medical School. He expressed the belief that a "complete mastoidectomy," with exenteration of all accessible cells, should be done. In his experience the zygomatic and tip cells were those most often incompletely removed.

MORTALITY

Among 298 patients (3 per cent) operated on for acute mastoiditis there were 9 deaths. All these deaths were caused by meningitis or sepsis or both. The term "sepsis" as used here connotes a well defined clinical picture of septicemia, though in a few of the cases micro-organisms

TABLE 4.—*Mortality in Entire Series*

	Cases	Percentage
Rawlins and Reich [6]	403	4.8
Coates, Ersner and Persky [9a]	644	4.5
Boyd [13]	188	4.2
Boies [1]	240	4.0
Kafka [14] (collected)	1,385	5.8
Tomb [4b] (private)	100	1.0
Present series	298	3.0

were not grown in blood cultures. Some patients might have had positive blood cultures but died before repeated cultures could be made. It is of interest to compare the mortality in the present series with the published reports shown in table 4.

With the exception of that reported by Tomb,[4b] the mortality rate in the series to be reported here is lowest. All the reports except the present one covered periods when sulfanilamide and its derivatives were not used. All the reports cited except Tomb's [4b] were from charity services. Examination of the data pertaining to the complicated cases

11. Lillie, H. I.: The Effect of Otitis Media on Patients Previously Operated on for Mastoiditis, Arch. Otolaryng. **8**:183-184 (Aug.) 1928.

12. Lacy, N. E.: Recurring Mastoiditis, Ann. Otol., Rhin. & Laryng. **42**:526-532 (June) 1933.

13. Boyd, A. A.: Report on One Hundred and Eighty-Eight Consecutive Cases of Acute Otitis Media with Mastoiditis in Which the Schwartze Operation Was Performed, J. Laryng. & Otol. **36**:217-228 (May) 1921; cited by Rawlins and Reich.[6]

14. Kafka, M. M.: Mortality of Mastoiditis and Cerebral Complications with Review of 3225 Cases, Laryngoscope **47**:790-822 (Oct.) 1935.

indicates a possible reason for the lower mortality in the series reported here.

COMPLICATIONS: SEPSIS AND MENINGITIS

In computing the mortality figures in this study, such complications as facial paralysis, postauricular fistula and other abnormal conditions not threatening to life were not considered. Sepsis and meningitis were the two potentially fatal complications encountered. One patient had a cerebellar abscess but also had septicemia and meningitis. Three had empyema resulting from septicemia. The incidence of sepsis is seen in table 5.

In 13 of the 16 cases of sepsis clinical indications of the condition were present when the patient was admitted to the hospital. In 3 of

TABLE 5.—*Data Pertaining to Sepsis Complicating Acute Surgical Mastoiditis*

Patients with positive blood cultures	13
Beta hemolytic streptococci	12
Gamma streptococci	1
Patients without positive blood cultures but with a well established clinical picture of sepsis	3
Death within these groups	1 (6.2%)*

* This patient never had a positive blood culture.

TABLE 6.—*Mortality from Sepsis*

	Percentage
Boies,[1] University of Minnesota	19
Meltzer,[15] Massachusetts Eye and Ear Infirmary (proved cases)	21.7
Present series	6.2

the cases they followed mastoidectomy. In 13 cases the lesion causing the sepsis was lateral sinus thrombophlebitis, in 2 cases acute mastoiditis and in 1 case perisinus abscess. The mortality from sepsis in this and other series is indicated in table 6.

The general and surgical management in the cases in these three series have been similar except that chemotherapy was added to the therapeutic measures in the present series. The decrease in the death rate is truly remarkable and naturally gratifying.

SURGICAL MANAGEMENT OF OTITIC SEPSIS

A brief discussion of the surgical management of otitic sepsis follows. No routine series of procedures has been followed in all cases of sepsis.

15. Meltzer, P. E.: Treatment of Thrombosis of the Lateral Sinus, Arch. Otolaryng. **22**:131-142 (Aug.) 1935.

The treatment has been suited to each case. This is in accordance with the views of others.[16]

In 1920 Rott [16d] reviewed the situation. He said that thirty years earlier Horsley had advocated ligation of the jugular vein only after sinus infection was proved by the presence of metastatic infections. He quoted Barck, of St. Louis, as a proponent of the opposite view. Barck is said to have advocated primary ligature of the jugular vein in all cases of sinus thrombosis. Between these two extremes Rott placed the views of many noted otologists, including Politzer, Koerner, Bezold, Allport, Whiting, Macewen, Loeb and Phillips. Rott's own opinion was that the treatment should be made to fit the case at hand. This point of view has been propounded by Lillie [17] and by Dixon,[18] who have decried the routine practice of ligation of the internal jugular vein and ablation of the sigmoid sinus. The crux of the matter seems to have been reached by Ballance,[19] who said, "All true and lasting surgical practice is based on pathology and when once pathology of an affection is clearly appreciated, divergence of view as to its treatment ought to disappear." Further discussion of the methods employed at University Hospitals follows.

If the clinical picture of sepsis was well established when the mastoidectomy was done, the sigmoid sinus was widely uncovered and inspected. If thrombosis seemed probable and obliteration of the sinus was to be carried out, the procedure in the more recent cases has been to ligate the homolateral internal jugular vein before proceeding with the sinus ablation. This sequence has been approved by Mosher.[20] Ligation of the jugular vein is thought to be a rational therapeutic procedure when combined with surgical closure of the sigmoid portion of the lateral sinus, with the consequent obliteration of side channels through which pathogenic micro-organisms might enter the general circulatory system. Necrotic material is removed from the sinus by suction. Perisinal abscess, with thick granulations on the sinus, has been seen many times when sepsis was not present. Exposure of the sinus until normal dura surrounded the granulations has been the usual procedure with this condition. Sepsis has not generally been considered a surgical

16. (*a*) Lillie, H. I.: Recent Clinical Observations in Involvement of the Blood Stream in Otitic Disease, Ann. Otol., Rhin. & Laryng. **37**:388 (March) 1928; (*b*) Suppuration of the Temporal Bone Accompanied by Infection of the Blood Stream, Arch. Otolaryng. **18**:630-642 (Nov.) 1933; (*c*) The Surgery for Sepsis of Otitic Origin, in Kopetzky, S. J.: Surgery of the Ear, New York, Thomas Nelson & Sons, 1938, chap. 9. (*d*) Rott, O. M.: Concerning the Question of Jugular Ligation in Sinus Thrombosis, Ann. Otol., Rhin. & Laryng. **29**:820-828 (Dec.) 1920.

17. Lillie, footnote 16 *a*, *b* and *c*.

18. Dixon, O. J.: A Departure in the Management of Infections of the Sigmoid Sinus, Arch. Otolaryng. **2**:453-457 (Nov.) 1925.

19. Ballance, cited by Lillie.[16c]

20. Mosher, H. P., in discussion on Boies.[1]

emergency. When its presence has been definitely established, however, operation has not been delayed for fear that metastatic infections would be set up. The growth of micro-organisms from the patient's blood has not been considered essential to the establishment of the diagnosis of otitic sepsis.

The jugular compression test for patency of the sigmoid sinus, described by Tobey and Ayer,[21] has given inconsistent results in the cases of this series just often enough to make the University Hospitals staff uncertain of its value. Probably its application has not always been perfect on account of inexperience on the part of the assistant, as Meltzer [15] suggested. Anatomic peculiarities of the sigmoid sinus, as described by Sutherland [22] and by Hilding,[23] may have been factors in

TABLE 7.—*Data Pertaining to Meningitis*

Patients with positive cerebrospinal fluid cultures		10
Beta hemolytic streptococci	5	
Nonhemolytic streptococci	3	
Pneumococci type III	2	
Patients without positive cerebrospinal fluid cultures		4
Deaths within both groups		3 (21.3%)
Deaths in group with positive cultures		3 (30%)

TABLE 8.—*Mortality from Meningitis*

	Mortality
Rawlins and Reich [6] (Stanford Service, San Francisco Hospital)	75%
Coates, Ersner and Persky [9a] (Medico-Chirurgical Hospital and the University of Pennsylvania Hospital, Philadelphia)	58%
Boies [24] (Massachusetts Eye and Ear Infirmary)	100%
Loenholdt [25] (University of Minnesota; untreated with sulfanilamide or any of its derivatives)	100%
Tripoli [26]	91.6%
Gray [27]	97.0%
Present series (as a whole)	21.3%
Present series (only patients with positive cerebrospinal fluid cultures)	30.0%

21. Tobey, G. L., Jr., and Ayer, J. B.: Dynamic Studies on the Cerebrospinal Fluid in the Differential Diagnosis of Lateral Sinus Thrombosis, Arch. Otolaryng. **2**:50-57 (July) 1925.

22. Sutherland, J. M.: Otitic Sinus Thrombosis, Arch. Otolaryng. **27**:1-34 (Jan.) 1938.

23. Hilding, A.: False Response to Jugular Compression (Tobey-Ayer) Test Due to Anomaly of the Lateral Sinus, Arch. Otolaryng. **26**:143-145 (Aug.) 1937.

24. Boies, L. R.: Lateral Sinus Thrombosis: Review of One Hundred and Eighty-Four Cases, Ann. Otol., Rhin. & Laryng. **41**:227-237 (March) 1932.

25. Loenholdt, E. H.: Otogenic Meningitis, Staff Meet. Bull., Univ. of Minnesota Hosp. **11**:35-44, 1939.

26. Tripoli, C. J.: Bacterial Meningitis: Comparative Study of Various Therapeutic Measures, J. A. M. A. **106**:171-177 (Jan. 18) 1936.

27. Gray, H. J.: Streptococcic Meningitis: Report of a Case with Recovery, J. A. M. A. **105**:92-95 (July 13) 1935.

some of the cases. Both of these possible sources of error were mentioned in the original paper by Tobey and Ayer.[21] At any rate, we have encountered instances in which patency was indicated and complete thrombosis found at operation immediately afterward. The opposite conditions have also been seen.

MENINGITIS

The potential danger of otogenic meningitis also has been reduced. Data relative to this are shown in table 7.

In 3 patients meningitis and sepsis occurred together. All these patients had organisms in both the blood and the spinal fluid, and all died.

Comparison of the results in a series of cases of otogenic meningitis in which treatment with sulfanilamide was not used with the results obtained in the present series shows that a distinct gain has been made in the therapy of this disease. In table 8 these results are listed.

A succinct statement concerning the progress in the therapy of otogenic meningitis caused by beta hemolytic streptococci is found in the comment of Schenck [28] on the reports of Gray [27] and of Trachsler.[29] In 1938 Schenck [28] said, "The number of recoveries from streptococcic meningitis since sulfanilamide has been available now exceeds the total number from 1901 to 1937."

THERAPY WITH SULFANILAMIDE AND ITS DERIVATIVES

From a consideration of the material just presented it is apparent that the number of deaths from potentially lethal complications has been reduced. This reduction must have resulted from the use of sulfanilamide and its derivatives, because no other material change in the mode of treatment has been made since the report of an exactly comparable series.[1]

A brief comment on the way in which sulfanilamide and its derivatives are used at University Hospitals may be of interest. Patients with otitis media are seldom seen at the hospital when the disease is in an early stage. Therefore, this discussion of therapy with sulfanilamide and its derivatives applies mainly to the postoperative period. In this series there were only 2 cases in which members of the hospital staff had control of the therapy early in the course of the disease. Both

28. Schenck, H. P.: Use of Sulfanilamide in Otolaryngology, Arch. Otolaryng. **28**:698-747 (Nov.) 1938.

29. Trachsler, W. H.; Frauenberger, G. S.; Wagner, C., and Mitchell, A. G.: Streptococcic Meningitis with Special Emphasis on Sulfanilamide Therapy, J. Pediat. **11**:248-269 (Aug.) 1937.

patients were hospitalized less than one week after the onset of earache. Material from the ear of each yielded beta hemolytic streptococci on culture. Surgical mastoiditis developed in both patients despite adequate sulfanilamide therapy. I do not generalize from those 2 instances. They prove, however, that mastoiditis caused by hemolytic streptococci can and does develop in the presence of seemingly adequate sulfanilamide therapy.

The value of sulfanilamide in the treatment of acute otitis media is difficult to assess. Woodward [30] reported 396 cases of mastoiditis over a ten year period. A sharp decline in frequency coincided with the introduction of sulfanilamide. On his graph a low point in 1934 equaled the postsulfanilamide level; this was prior to the use of the drug. His postsulfanilamide low level may have been due to other factors entirely. Horan and French [31] reported an incidence of 22.7 per cent for mastoiditis in 607 cases of acute otitis media untreated with sulfanilamide and 4.5 per cent in a series of 155 treated patients. On the other hand, Page [32] cited an incidence of mastoidectomy of 5.1 per cent in 836 patients who had myringotomy in 1934 and 37 per cent in a series of 1,029 patients treated with sulfanilamide in 1937.

Fisher,[33] reporting from the Johns Hopkins University, found a great diminution in the frequency of surgical mastoiditis in a group of patients treated with sulfanilamide as compared with an untreated control group. Whether such results will be constant or will vary from year to year only time can tell.

The toxic reactions and the masking effect of sulfanilamide have been widely discussed. Maybaum, Snyder and Coleman [34] advocated the use of the drug only in serious otitic complications, because of its masking effect. Rosen [35] reported a case in which masking was a factor. Bowers [36] found it "necessary at times to stop the drug in order to obtain the true

30. Woodward, F. D.: Sulfanilamide and Roentgen Ray Therapy for Acute Otitis Media and Mastoiditis, Laryngoscope **49**:572-579 (July) 1939.

31. Horan, V. G., and French, S. G.: Sulfonamide (Sulfanilamide) in Acute Mastoiditis, Brit. M. J. **2**:942-943 (Nov. 5) 1938.

32. Page, J. R., in discussion on Woodward.[30]

33. Fisher, G. E.: Sulfanilamide in the Treatment of Otitis Media, J. A. M. A. **112**:2271 (June 23) 1939.

34. Maybaum, J. L.; Snyder, E. R., and Coleman, L. L.: Sulfanilamide Therapy for Otogenous Infections, with Special Reference to Masking of Clinical Course, Arch. Otolaryng. **30**:557-575 (Oct.) 1939.

35. Rosen, S.: Acute Mastoiditis Masked by Sulfanilamide, Laryngoscope **50**: 198-199 (Feb.) 1940.

36. Bowers, W. C.: Observations on Seven Hundred and Ninety-Three Cases of Acute Purulent Otitis Media with Chemotherapy in Three Hundred and Ninety-Six Cases, J. A. M. A. **115**:178-181 (July 20) 1940.

picture, as the drug cures the middle ear while progressive bone destruction is taking place in the mastoid." An exhaustive review of the literature was published by Schenck [28] in 1938. Those interested in the mechanism of action or in the toxic effects are referred to Schenck's paper.

Harkness [37] recently expressed a seemingly reasonable opinion on the use of sulfanilamide. He directed attention to the inconsistency of condemning

> . . . a therapeutic measure of acknowledged efficiency for calamitous complications for use in treatment of the minor conditions that lead to these serious complications. . . . Rather its usefulness should be acknowledged, with precautions against the potentialities for harm and a realization that temporary masking of symptoms does not mean final recovery. These considerations should permit the use of the drugs to prevent complications.

Sulfanilamide was the drug used in most of the cases of this series. Occasionally sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) or sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) was used. In 57 patients (19.4 per cent) one of these three compounds was used. Twenty-eight cases were uncomplicated and 29 were complicated. The usual indication for the administration of sulfanilamide or one of its derivatives in an uncomplicated case was postoperative fever. The average number of days of therapy in the complicated cases was eight, and in the uncomplicated ones, nine and one tenth. Blood levels were not taken in the earlier cases. In the later ones, comprising 23, the average level of 11.5 mg. per hundred cubic centimeters was above the accepted adequate therapeutic range of 5 to 10 milligrams. Not enough patients were treated with sulfapyridine and sulfathiazole to make any generalizations possible.

It now seems to be clearly established that sulfathiazole should not be used for pneumococcic mastoiditis with meningitis. After reviewing the English literature on pneumococcic meningitis, Gilmore and Sacks [38] said, "The varied chemotherapeusis of pneumococcic meningitis has been replaced by treatment with a single chemical, sulfapyridine, with beneficial results." Sulfathiazole does not enter the spinal fluid in sufficient concentration to be effective.[39] The administration of sulfapyridine and specific antipneumococcus serum [40] is indicated.

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37. Harkness, C. F.: Complications of Surgical Treatment of Acute Mastoiditis, Arch. Otolaryng. **32**:915-926 (Nov.) 1940.

38. Gilmore, G. B., and Sacks, P.: Pneumococcic Meningitis: A Review of the English Literature, Arch. Otolaryng. **32**:1007-1022 (Dec.) 1940.

39. Sadusk, J. F., and Nielsen, A. E.: Use of Sulfathiazole in Staphylococcic Meningitis with Recovery, J. A. M. A. **116**:298-300 (Jan. 25) 1941.

40. Sadusk, J. F., Jr.; Blake, F. G., and Seymour, A.: Observations on the Absorption, Excretion, Diffusion and Acetylation of Sulfathiazole in Man, Yale J. Biol. & Med. **12**:681-696 (July) 1940. Spink.[2]

MISCELLANEOUS COMPLICATIONS

Paralysis of the facial nerve was encountered six times. In 5 of the 6 patients it was present on admission to the hospital. In half of the cases complete recovery occurred after simple mastoidectomy. A nerve graft by the method of Ballance and Duel [41] was performed on 1 patient.

Empyema occurred three times in cases of sinus thrombosis. In each instance beta hemolytic streptococci were cultured from the pleural fluid. In 2 of the patients the aspiration of pleural fluid, together with chemotherapy, effected a cure. One patient had surgical pleural drainage before being cured. This complication apparently is seen less frequently now than it was many years ago. Macewen [42] regarded pulmonary difficulties as not uncommon with sinus thrombosis. In 1931 Lillie [43] reported the occurrence of disease of the sigmoid sinus in 88 of 933 cases in which mastoidectomy had been done. Pleural effusion or pulmonary infarction developed in only 4 of the patients with sinus disease.

Petrositis with Gradenigo's [44] syndrome was encountered three times. In each case exenteration of the mastoid was sufficient to produce recovery. The palsy of the abducens nerve disappeared completely in each instance. Apicectomy was not necessary in any case. The number of perilabyrinthine petrous cell tracts opened during mastoidectomy has been high. Cases in which this has occurred have not been classified as cases of petrositis, although the tracts are in the base of the petrous pyramid.

Other complications encountered included panophthalmitis, pyarthrosis, osteomyelitis, erysipelas, parotid abscess, serous labyrinthitis and otitic hydrocephalus. Otitic hydrocephalus is sufficiently rare and the case encountered was so typical that discussion seems merited.

In 1931 Symonds [45] proposed the term "otitic hydrocephalus" for that condition in which increased intracranial pressure is the only com-

41. Duel, A. B., and Tickle, T. G.: The Surgical Repair of Facial Nerve Paralysis: A Clinical Presentation, Ann. Otol., Rhin. & Laryng. **45**:3-27 (March) 1936.

42. Macewen, W.: Pyogenic Infective Diseases of the Brain and Spinal Cord, New York, Macmillan & Co., 1893, p. 266.

43. Lillie, H. I.: Pulmonary Complications Associated with Thrombosis of the Sigmoid Sinus: Report of a Case, Ann. Otol., Rhin. & Laryng. **40**:30-33 (March) 1931.

44. Gradenigo, G.: Sur un syndrome particulier de complications endocraniennes otitiques; paralysie de l' abducteur d' origine otitique, Ann. d. mal. de l' oreille, du larynx **30**:120, 1904, cited by Almour, in Kopetzky, S. J.: Surgery of the Ear, New York, Thomas Nelson & Sons, 1938, p. 331.

45. Symonds, C. P.: Otitic Hydrocephalus, Brain **54**:55-71 (April) 1931.

plication of aural infection. The composition of the spinal fluid was normal, according to all the usual laboratory tests, but the pressure was much above the normal level. The condition had previously been called "serous meningitis," "ependymitis serosa" and other names implying a knowledge of the causation or of the pathologic changes greater than actually existed. In 1924 Adson [46] reported 3 cases as cases of "pseudobrain abscess." In 1913 Passot [47] pleaded for a classification of the nonpurulent intracranial complications of disease of the temporal bone on the basis of the chemical and microscopic examination of the cerebrospinal fluid. He distinguished between two conditions. In one, the clinical symptoms are those of mild meningitis, and there is an increase of cells and protein in the cerebrospinal fluid. In the second type, there is increased pressure, but the fluid is normal. Passot thus established the occurrence of intracranial hypertension without brain abscess and with normal cerebrospinal fluid.

Symonds' [45] diagnostic criteria were: 1. The condition usually occurs in children or adolescents. 2. The patient feels well between headaches. 3. Papilledema is constant and usually is measurable; exudates usually are present and hemorrhages occur in the ocular fundi. 4. There are no cerebral localizing signs, but palsy of the abducens nerve on the side of the aural lesion is often present. 5. The spinal fluid is clear, with pressure often over 300 mm. of water, abundant in quantity and with no excess protein or cells. 6. Recovery usually occurs.

An extensive review of the confused and voluminous literature on the subject was published by Williams [48] in 1938. Case reports by various authors have appeared.[49]

The essence of the matter seems to be as follows: Increased intracranial pressure with normal cerebrospinal fluid can occur as a complication of aural lesions. No space-occupying cerebral lesion can be demonstrated. There may be a communicating hydrocephalus or internal hydrocephalus. In the latter condition surgical removal of fluid from the ventricle is necessary for cure (Williams,[49a] case 3, for example).

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46. Adson, A. W.: Pseudobrain Abscess, S. Clin. North America **4**:503-512 (April) 1924.

47. Passot, R.: Méningites et états méningés aseptiques d' origine otitique, Thesis, Paris, no. 247, Paris, G. Steinheil, 1913; cited by Symonds.[45]

48. Williams, H. L.: The Underlying Factors Concerned in Otitic Hydrocephalus, Ann. Otol., Rhin. & Laryng. **47**:670-714 (Sept.) 1938.

49. (*a*) Williams, H. L.: Otitic Hydrocephalus, Arch. Otolaryng. **25**:632-652 (June) 1937. (*b*) Smith, C. H.: Symposium on Facial Nerves: II. Otitic Hydrocephalus with Report of Case, Laryngoscope **44**:931-934 (Dec.) 1934. (*c*) Mygind, H.: Benign Forms of Otogenic Meningitis, J. Laryng. & Otol. **37**:597-612 (Dec.) 1922; cited by Smith.[49b] (*d*) Cairns, H.: Abscess of Brain, J. Laryng. & Otol. **45**:385-397 (June) 1930; cited by Smith.[49b] (*e*) Carmack, J. W.: The Relation of Increased Intracranial Pressure to Disease in the Pneumatic Spaces, Ann. Otol., Rhin. & Laryng. **42**:364-371 (June) 1933. Symonds.[45] Adson.[46]

The causation and pathogenesis are unknown. The course of the patient observed at University Hospitals and the surgical findings in the case are summarized. They seem to be typical of the communicating type of the disease.

REPORT OF A CASE

N. M., aged 19 years, was admitted to the hospital on June 10, 1940 for treatment of an acute exacerbation of chronic otitis media on the right side. The otitis had followed swimming three years before and had recurred many times. For three weeks just before admission the girl had never been free from fever, and at one time her temperature had reached 106 F. During the twenty-four hours following admission no signs of sepsis occurred. On June 11 radical mastoidectomy was done on the right side. There was extensive necrosis of bone, exposing areas of the sigmoid sinus and of the dura of the middle fossa. These areas were covered with thick granulation tissue. Normal dura was surgically exposed around the granulation tissue. Forty-eight hours after operation septic temperature appeared, with a daily rise to 104 F. On June 16 ligation of the jugular vein was done. The vein was collapsed above the junction with the common facial vein. When the sigmoid sinus was incised through the granulation bed covering it, about 4 cc. of thin gray pus flowed out. Free bleeding was obtained from the superior end but none from the inferior end. The sinus was compressed above with packing but was packed within the lumen below.

The patient responded well to the ligation. Her subsequent course was uneventful except for an abscess in the neck wound and occasional headaches. On the twenty-seventh day after admission, when she was about to be sent home, she complained of double vision on gazing to the right.

Ophthalmologic consultants demonstrated normal vision and bilateral papilledema of 4 diopters, with incomplete paralysis of the right abducens nerve. No neurologic signs could be found to indicate the presence of a space-taking cerebral lesion. A general examination, including studies of the blood, urinalysis and serologic tests for syphilis, revealed normal conditions. The spinal fluid was under a pressure greater than 600 mm. of water (over the manometer top). This fluid contained no cells but contained 69 mg. of sugar, 25 mg. of protein and 705 mg. of chlorides per hundred cubic centimeters. The colloidal gold curve was normal and tests for syphilis gave negative results.

On July 16 Dr. W. T. Peyton, of the neurosurgical staff, demonstrated normal filling of the cerebral ventricles with air. A routine of spinal drainage and intravenous administration of a hypertonic solution of dextrose was begun. The spinal fluid was normal in composition. After a week it was apparent that the spinal fluid pressure was not being lowered by the treatment. The patient was perfectly comfortable, and her vision remained nearly normal. She was discharged from the hospital on August 17 and was followed closely in the outpatient department thereafter.

Her last visit occurred on October 1. Her vision was then:

Right eye 20/30 distant
14/21 near (Americal Medical Association charts)

Left eye 20/20 distant
14/14 near

Both optic disks were elevated between 1 and 2 diopters. She was perfectly comfortable, being free of headaches, and easily able to do her work as a restaurant cook.

POSTOPERATIVE HEARING

The general opinion among otologists is that in previously normal ears hearing loss after simple or complete mastoidectomy is usually negligible. Since the advent of the audiometer measurement of the postoperative hearing level has been more accurate. No published reports of such measurements of postoperative hearing have been encountered. The following charts and discussion are therefore appended. The post-

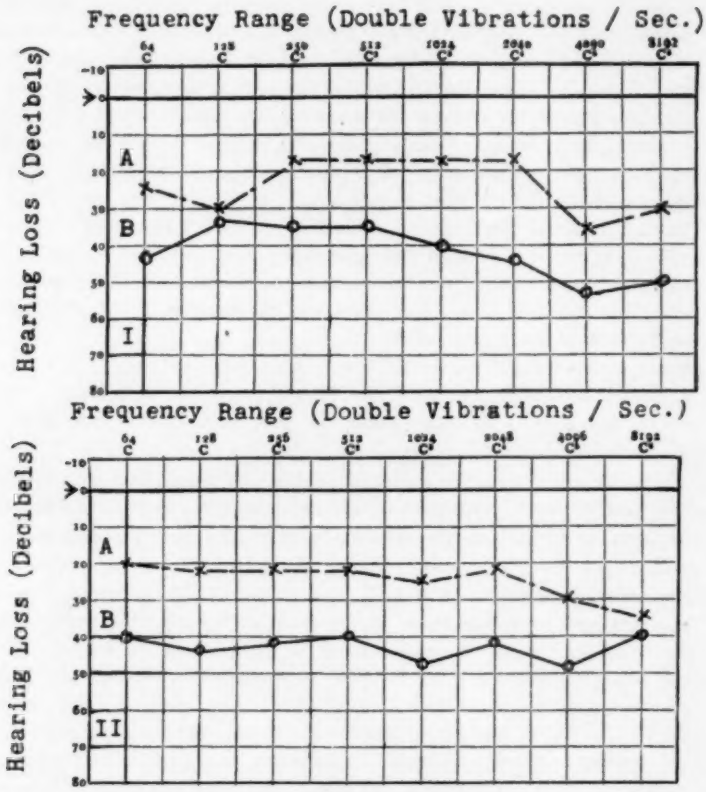

Chart 2.—Preoperative and postoperative hearing level of (*I*) 55 patients who had acute surgical mastoiditis without previous otitis media or mastoiditis, and (*II*) 7 patients who had acute surgical mastoiditis and had previously had an incomplete mastoidectomy. *A* indicates the postoperative and *B* the preoperative levels.

operative hearing measurements would be of more value if the hearing of these same patients could have been measured before their otitis media began.

Chart 2 *I* shows the preoperative and postoperative threshold hearing levels of 55 patients on whom complete mastoidectomy was performed. These patients had no history of chronic otitis media or chronic mastoiditis. Their audiograms, made under as nearly identical conditions

as possible, should be strictly comparable. Only patients who had both preoperative and postoperative audiograms were used in the calculation. Their audiograms were made at varying postoperative intervals. In all cases, however, the preoperative level was measured within a few days of the operation. It is probable, therefore, that the postoperative curve is lower than it would have been if all audiograms had been made after a long postoperative interval. In a clinic such as that at University Hospitals this is a difficult factor to control unless one disregards the patient's expense and time.

The preoperative level is seen to be much lower than the postoperative one. In many cases the postoperative level closely approximated the normal. The average, however, was as shown. It is pertinent here to

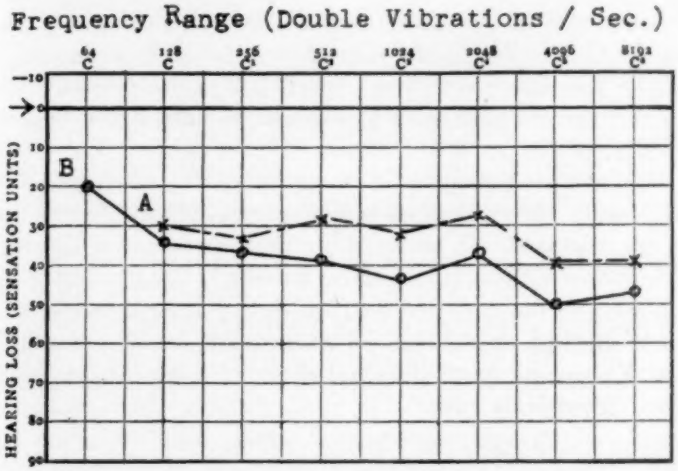

Chart 3.—(*A*) the postoperative and (*B*) the preoperative hearing level of patients who had acute exacerbations of chronic mastoiditis and who were treated by complete mastoidectomy.

consider the statement of Bunch,[50] who cautioned one to remember ". . . that clinical tests with modern audiometers can be reproduced in repeated tests with an accuracy of from five to seven and a half decibels . . ."

In chart *2 II* are shown the hearing levels of those patients on whom incomplete mastoidectomy had been performed prior to a second operation on the mastoid bone. The group is small, comprising *7* patients. Their postoperative hearing was almost as good as that of patients whose ears had not been operated on previously.

50. Bunch, C. C.: Usable Hearing, Ann. Otol., Rhin. & Laryng. **49**:359-367 (June) 1940.

In chart 3 are seen the hearing levels of the patients having chronic mastoiditis who were treated by complete or by simple mastoidectomy rather than by radical mastoidectomy. While these patients have poorer hearing than those of either of the first two groups, their postoperative hearing is much better than the preoperative level.

SUMMARY

A report on some aspects of acute surgical mastoiditis is presented. Cases occurring over a ten year period are analyzed as to annual, seasonal and age incidence. No change in incidence has occurred since sulfanilamide and its derivatives were introduced.

Two hundred and ninety-six cases of acute surgical mastoiditis occurring from 1936 to 1940 are analyzed in detail. The mortality in the 296 cases was 3 per cent. All deaths were caused by sepsis, meningitis or both. The death rate among the patients having these complications was lower than that reported in many previous studies. The reduction seemed to be due to the use of sulfanilamide or one of its derivatives.

The surgical management of otitic sepsis at University Hospitals is discussed briefly. Miscellaneous complications, including otitic hydrocephalus, are discussed. In all types of cases postoperative hearing was found to be improved over preoperative hearing. The greatest improvement was seen in the ears operated on during a first attack of otitis media with surgical mastoiditis. Postoperative hearing among both patients with recurrent mastoiditis and those in whom the condition was chronic was surprisingly good.

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MELANOSIS OF THE INTERNAL EAR

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Deposits of pigment in the internal ear have been observed in mammals by a number of pathologists. Certain questions have arisen relative to the origin of the pigment, but its presence has been accepted as a commonplace fact without pathologic significance. In a study of 28 human membranous labyrinths, Wolff [1] reviewed the reports in the literature, including those of Corti,[2] Voltolini,[3] Lucae,[4] Waldeyer,[5] Retzius,[6] Alexander,[7] Siebenmann,[8] Ranvier,[9] Kolmer [10] and Maximow.[11] It is clear that she was impressed by Kolmer's statement that the amount of pigmentation in the region of the labyrinth is dependent on the general pigmentation of the animal and that therefore labyrinthine coloration is lacking in albinos. Wolff therefore questioned all reports in the literature in which pigment was stated to be absent and generally concluded that the animals used as subjects must have been white ones.

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1. Wolff, D.: Melanin in the Inner Ear, Arch. Otolaryng. **14**:195 (Aug.) 1931.

2. Corti: Recherches sur l'organe de l'ouïe des mammifères, Ztschr. f. wissensch. Zool., 1851, vol. 3.

3. Voltolini, R.: Anatomische und pathologisch-anatomische Untersuchungen des Gehörorganes, nebst 5 Sections-fällen, Virchows Arch. f. path. Anat. **18**:34, 1860; Sectionen von Schwerhörigen, ibid. **22**:110, 1861; Sectionsergebnisse bei Schwerhörigen und Taubstummen, ibid. **27**:159, 1863.

4. Lucae, A.: Anatomisch-Physiologische Beiträge zur Ohrenheilkunde, Virchows Arch. f. path. Anat. **29**:33, 1864.

5. Waldeyer, in Stricker, S.: Handbuch der Lehre von den Geweben des Menschen und der Tiere, Leipzig, W. Engelmann, 1871, p. 943.

6. Retzius, G.: Das Gehörorgan der Wirbeltiere, Stockholm, Samson & Wallin, 1881, vol. 1.

7. Alexander, G.: Das Labyrinthpigment des Menschen und der höheren Säugetiere, Arch. f. mikr. Anat. **58**:134, 1901.

8. Siebenmann: Das innere Ohr, in Bardeleben, K.: Handbuch der Anatomie des Menschen, Jena, Gustav Fischer, 1906, p. 308.

9. Ranvier, L.: Traité technique d'histologie, Paris, F. Lavy, 1889, p. 767.

10. Kolmer, W., Haut- und Sinnesorgane, in von Möllendorf, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1927, vol. 3.

11. Maximow, A. A.: A Textbook of Histology, completed and edited by W. Bloom, Philadelphia, W. B. Saunders Company, 1930.

A. A. Gray [12] stated that it would be interesting to know whether pigment is found more abundantly in the labyrinth in Negroes than in members of the white races. Guild [13] stated that Negroes have more pigment than white persons. He expressed the belief that it is difficult to state how much more, because there are very few "pure" Negroes.

Wolff stated the belief, founded on her researches, that the membranous labyrinth should be included, along with such structures as the retina and the choroid coats of the eye, the arachnoid of the brain and spinal cord and the epidermis, in the list of depots for branched melanophores. In human embryos, pigment does not appear in the ear as soon as it appears in the eye. Melanin was not found in the skin of human fetuses up to 6 months even among Negroes. In all Negroes, including stillborn infants, pigment is seen in the labyrinth. The age when it first appears is not yet known. Among white persons pigment is seen in this area more often in males than in females. Both Alexander and Wolff observed that in human beings the heaviest pigmentation is along the medial wall of the scala vestibuli and the interstices of the cribriform modiolus and that the pigment is contained in large branching anastomosing connecting tissue-like cells. In the animals examined, pigment occurred in the stria vascularis, if at all, and never in the interstices of the modiolus.

The extensive description of a case to be given presently seems justified, first, because of the rarity of the condition. As a matter of fact, in the literature at my disposal, I could not find a similar case recorded. Second, the case study offers additional light on the biology of melanomas.

REPORT OF A CASE

History.—E. S., a Negro girl 11 years of age, had when she was 3 years old some type of "inflammation of the brain," which the mother cannot describe with any further detail. Immediately after this illness, difficulties with speech were noted. When the child spoke, her mouth was drawn to the right. She never could walk in the normal way. In 1935 she had an accident. Two weeks prior to her admission to the hospital she complained of pain and weakness in the legs, so that she was forced to remain in bed. For the past month she had also noticed a diminution in vision and had vomited occasionally. Since February 1939 headaches had been present. She was never able to attend school. The mother of the child has syphilis, which was never treated.

Clinical Findings.—On admission to the hospital, Nov. 4, 1939, the following conditions were noted: The pupils were round and dilated, the response to light was sluggish and there was no reaction to accommodation. There were abducens paresis on both sides and proptosis of both eyes. The left palpebral fissure was larger than the right. Both tympanic membranes were normal. Facial paralysis

12. Gray, A. A.: The Labyrinth in Animals and Man, London, J. & A. Churchill, 1907, vol. 1, p. 34.

13. Guild, S.: Personal communication to the author.

of a central type was present on the left side. The tongue did not deviate, and the pharynx was hyperemic. There was choking of the disk of 2.5 to 3 diopters on both sides, with rigidity of the neck and definite weakness of both legs and arms. Coordination was good; the Babinski reflex on both sides was positive. Abdominal reflexes were normal. On November 6 examination of the blood showed 72 per cent hemoglobin, 5,500 leukocytes, 62 per cent polymorphonuclear cells, 36 per cent lymphocytes, 2 per cent monocytes and basophilic stippling of erythrocytes. The patient died on November 21. The premortem diagnosis was glioma of the left cerebellopontile angle.

Autopsy.—At autopsy, scattered porotic areas were found in the skull. There was diastasis of the sutures of the skull (diastasis cranii). The gyri of the brain were flattened, and the meninges were thinned to a great degree. The inferior and lateral surfaces of the cerebellum were covered with a firm grayish tumor mass. Close to the left cerebellopontile angle there was a firm tumor mass, in which numerous nerves were embedded. The lateral ventricles were dilated. There were moderate eccentric hypertrophy of the heart, with dilatation of all the chambers; passive hyperemia of the lungs, kidneys and liver; parenchymatous degeneration of the heart, lungs and kidneys, and stagnation catarrh of the stomach.

Microscopic Examination of the Tumor.—Sections of the tumor showed a network of longitudinal spindle cells, in the meshes of which were embedded polymorphonuclear cells, each with a large, regularly formed hypochromatic nucleus. The protoplasm of some of the spindle cells was loaded with brown or yellow kernels of pigment, which were found particularly close to the capillaries. Many of the round cells contained the same type of pigment. There were no transitional cells between the longitudinal and round cells. The tumor replaced the meninges and invaded the sulci of the cerebellum but did not invade the substance of the brain to any great extent. A diagnosis of "diffuse melanocarcinomatosis of the meninges of the cerebellum" was arrived at.

The two temporal bones were turned over to my laboratory for microscopic examination. Unfortunately, it was not possible to remove the entire temporal bones, so that only the internal ears were at my disposal, and the left internal ear was opened at autopsy. Nevertheless, both pyramids were microscopically dissected in serial section and stained with hematoxylin and eosin.

Microscopic Examination of the Temporal Bones.—(*a*) Left Internal Ear: In the internal meatus were numerous polymorphonuclear cells, some the size of lymphocytes and others larger. Only occasionally was there some pigment within these cells, but each of them contained a nucleus rich in chromatin. These cells piled up and lay partly between the large nerves of the internal meatus, occasionally invading the nerves. There is no doubt that they were cells of the melanoma, which were carried by the cerebrospinal fluid into the internal auditory meatus. The size of the internal meatus was normal, and if one disregards the infiltration mentioned earlier, the nerves and the vestibular ganglion may be said to have been normal.

Only a part of the cochlea was preserved. Corti's organ showed postmortem changes. Corti's membrane was torn as the result of the autopsy technic, but the spiral ganglion and spiral nerve were normal, as was the bony capsule of the cochlea. The scala tympani of the basilar portion was very narrow, and in its mesial and inferior angle there were remnants of embryonal connective tissue, which contained some pigment cells (fig. 1 *A*).

In the channels of the modiolus there was a considerable amount of pigment. This pigment filled the cells of the loose connective tissue which occupied the space between the walls of the blood vessels and the bony walls of the channels

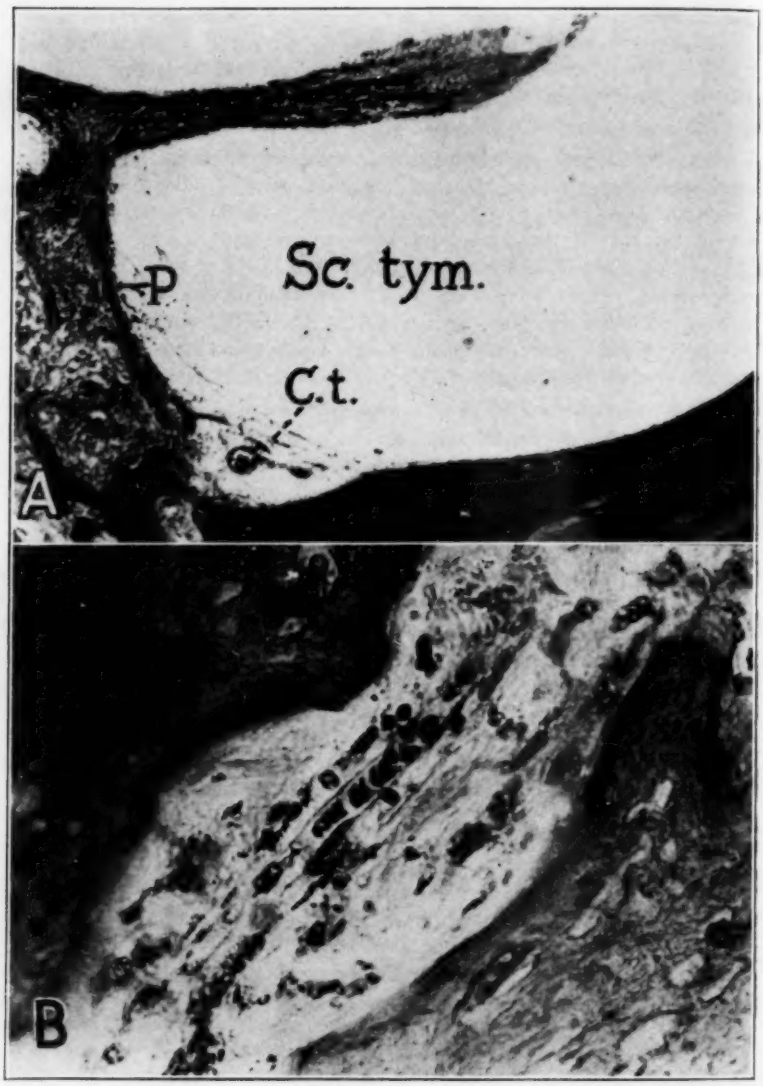

Fig. 1.—*A*, section through the basilar portion of the cochlea of the left side. Pigment (*P*) is found in the endosteum of the scala tympani (*Sc. tym.*) and the lamina spiralis ossea. The loose embryonal connective tissue (*C. t.*) also contains pigment. *B*, section through a marrow space in the bony septum of the modiolus (left side). There is pigment in the cells of the loose connective tissue which occupies the space between walls of the blood vessels and the bony walls of the channels of the modiolus.

of the modiolus (fig. 1 *B*). Some of these cells had a ramified appearance, so that they appeared like chromatophores. Other cells were so filled with pigment that the nucleus could not be seen. The same pigment was also found in great quantity between the fibers of the spiral nerve (fig. 2 *A*), in the endosteum of

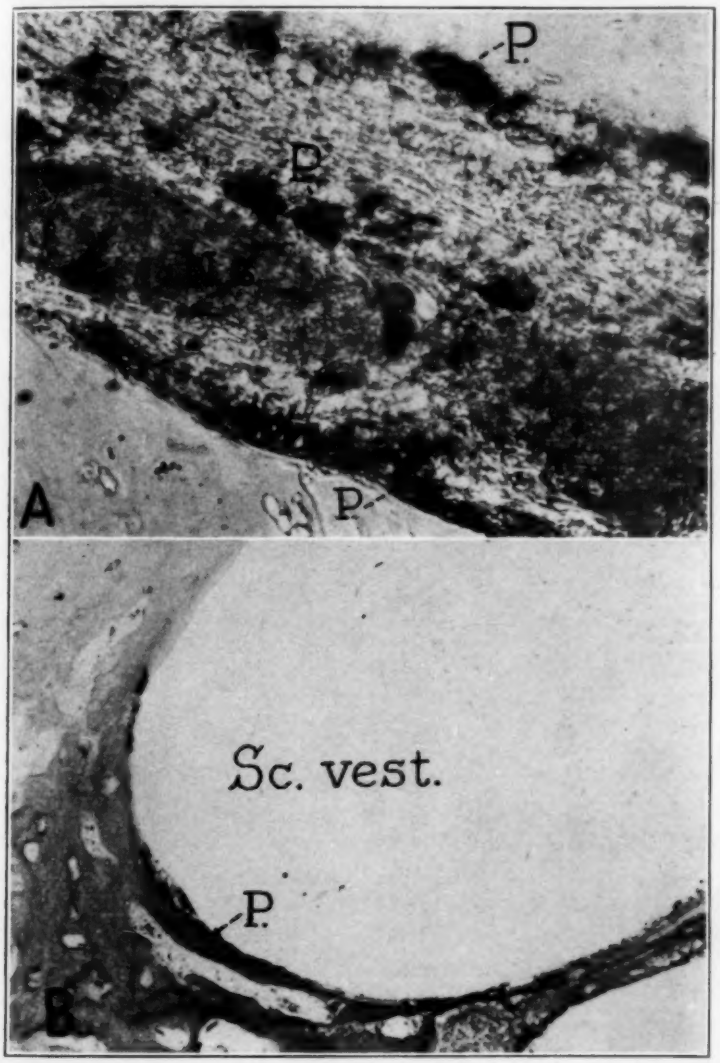

Fig. 2.—*A*, section through the lamina spiralis of the middle turn of the cochlea on the left side, which was made from an unstained specimen. Some of the cells containing pigment (*P*) have a ramified appearance, so that they resemble chromatophores. The pigment is also observed in a great quantity between fibers of the spiral nerve (*N. s.*). *B*, section through the basilar portion of the cochlea on the left side. Pigment (*P*) is observed in the endosteum of the scala vestibuli (*Sc. vest.*), as well as in the endosteum of the lamina spiralis ossea.

the scalae (fig. 2 *B*) and in the "tympanale Belegschicht" of the cochlea. Only occasionally, a single pigment cell was seen in the membrane of Corti, in the spiral ligament (fig. 3) and in the stria vascularis. The pigment cells frequently followed the course of the blood vessels of the capillaries; however, the walls of the capillaries apparently did not contain pigment. The dura contained no pigment, but there were many calcified bundles of connective tissue and calcified capillaries within the dura above the superior angle of the pyramid. The pigment observed was of a yellow or brown color in the hematoxylin-eosin stained specimen. When the kernels of pigment were firmly packed together, the color was black. The same color was observed also in those specimens which were unstained. The reaction with prussian blue was negative, and there was discoloration of the pigment with hydrogen peroxide, proof that the pigment was not derived from blood but was a melanin deposit.

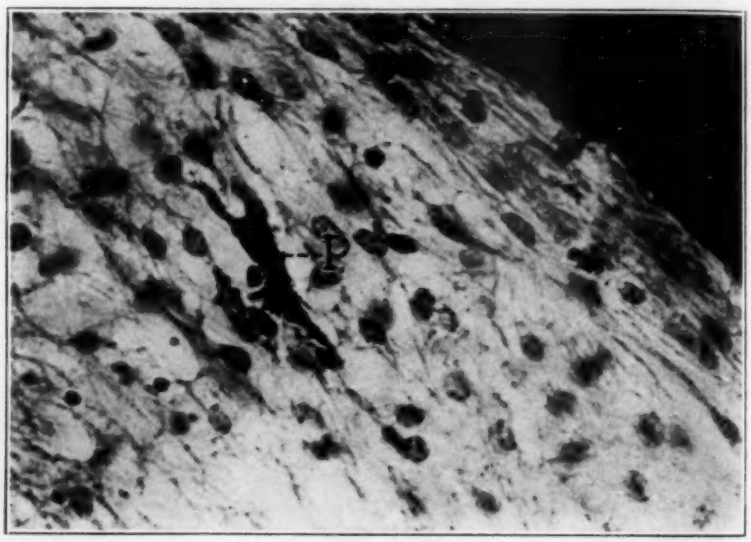

Fig. 3.—Pigmented (*P*) connective tissue cells in the spiral ligament.

(*b*) Right Middle Ear: The mucous membrane was thickened and contained coarse connective tissue and considerable pigment. The ossicles and drum membrane were not present. In the membrana secondaria there was a great deal of pigment, particularly at the site of the mesial insertion of the membrane (fig. 4). The oval window was normal and did not contain pigment.

(*c*) Cochlea: There was pigment in almost all the channels of the modiolus. The periosteum of these channels was thickened and showed hyaline degeneration in many areas. Within this hyaline connective tissue one observed the pigment cells as well as free kernels of pigment. Some marrow channels were entirely filled with hyaline connective tissue and pigment. There was slight atrophy of the spiral nerve and spiral ganglion of the basilar portion. The spiral ganglion of the basilar portion contained only one single cavity, which was not divided by bony septums, and thus resembled the ganglion in its embryologic state (fig. 5).

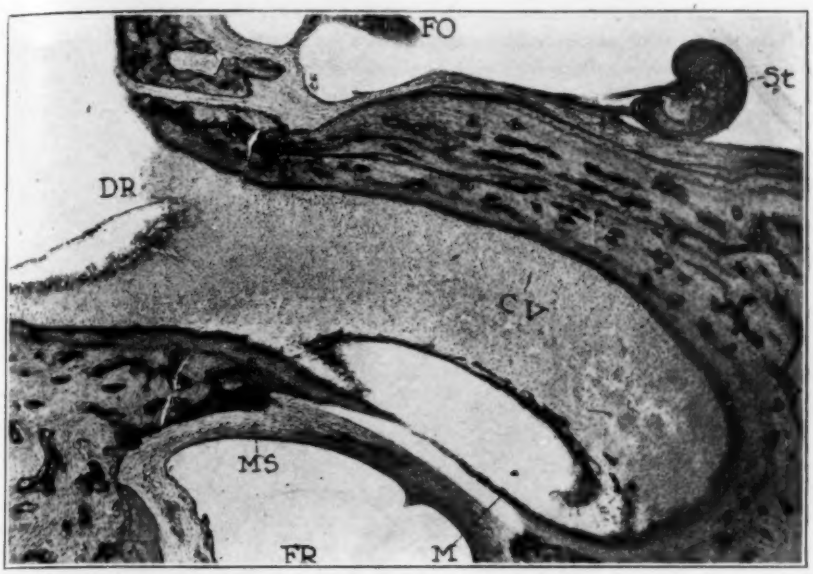

Fig. 4.—Section through the middle ear and cochlea of the right side. *FO* is the oval window; *St*, the stapes; *CV*, the caecum vestibulare; *DR*, the ductus reuniens; *FR*, the round window; *M*, the lamina spiralis ossea secondaria, and *MS*, the membrana tympani secundaria, containing deposits of pigment.

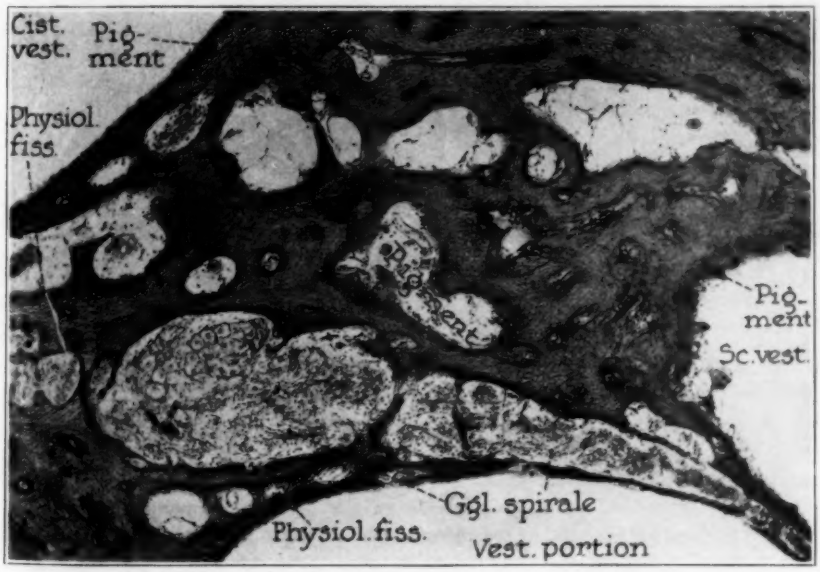

Fig. 5.—Section through the vestibular ganglion on the right side. Note the pigment in all of the marrow spaces and in the channels of the blood vessels. *Cist. vest.* is the cisterna vestibuli. Two physiologic fissures (*Physiol. fiss.*) are observed.

This cavity contained a considerable amount of sclerotic connective tissue, in which there were some small accumulations of ganglion cells, the number of these cells being less than normal. The cochlea was very well preserved. Corti's organ showed slight postmortem changes but did not contain pigment. The same was true of the stria vascularis and of Corti's membrane.

(*d*) Vestibulum: There was some pigment in the wall of the ductus reuniens, in the endosteum of the bony vestibule, particularly in its mesial wall, in all small

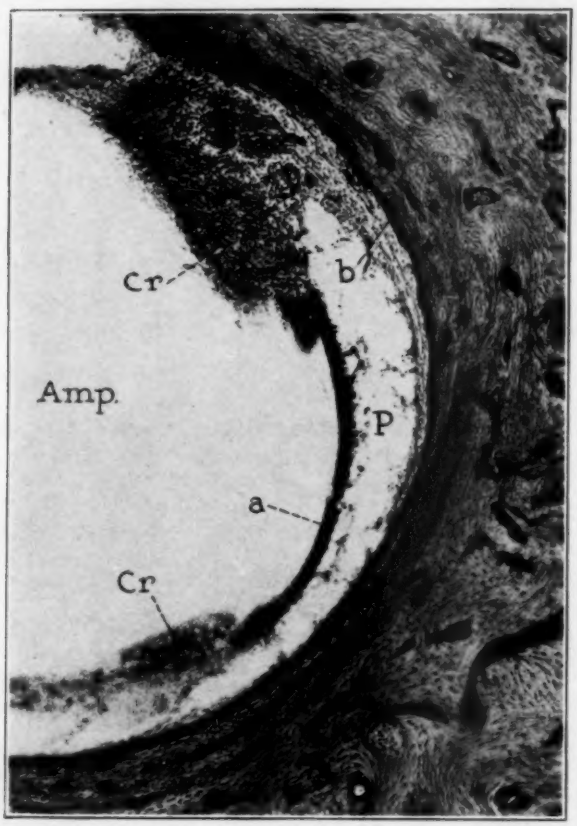

Fig. 6.—Section through the right sagittal semicircular canal. *Amp.* is the ampulla of this canal; *Cr,* the crista ampullaris, and *P,* the perilymphatic space. Pigment is seen in the membranous well of the ampulla (*a*) and in the endosteum of the ampulla (*b*).

marrow spaces and blood vessel spaces situated between the endosteal and the enchondral layer and in the periosteum of the ductus endolymphaticus. There was an absence of pigment in the macula utriculi and macula sacculi. There were two atypical epithelial spots in the sinus utricularis inferior, and both of them were definitely pigmented.

(*e*) Semicircular Canals: Some pigment was observed in the sagittal semicircular canal, close to the ampulla (fig. 6). Pigment was found also in the endosteum of the bony ampulla, in the small marrow spaces close to the endosteal

layer and in the perilymphatic tissue adherent to the membranous semicircular canal (fig. 7). The peak of the crista did not evidence pigment; however, the declinations were pigmented, conforming to the normal conditions. In the region

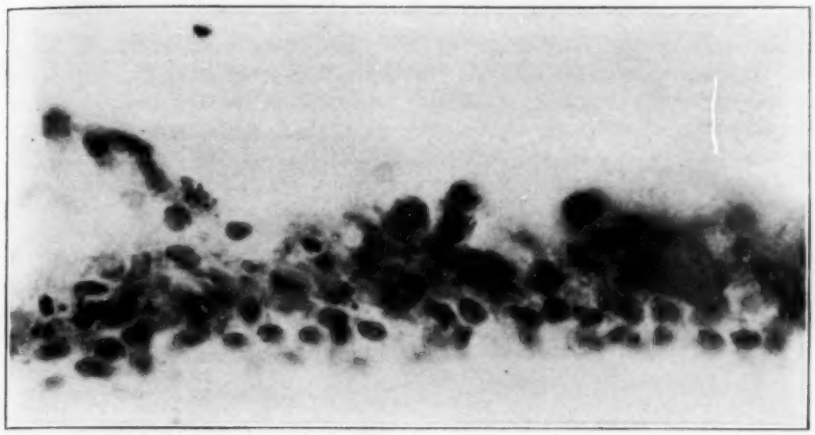

Fig. 7.—Section through the wall of the sagittal semicircular canal. Note granular pigment within the cells.

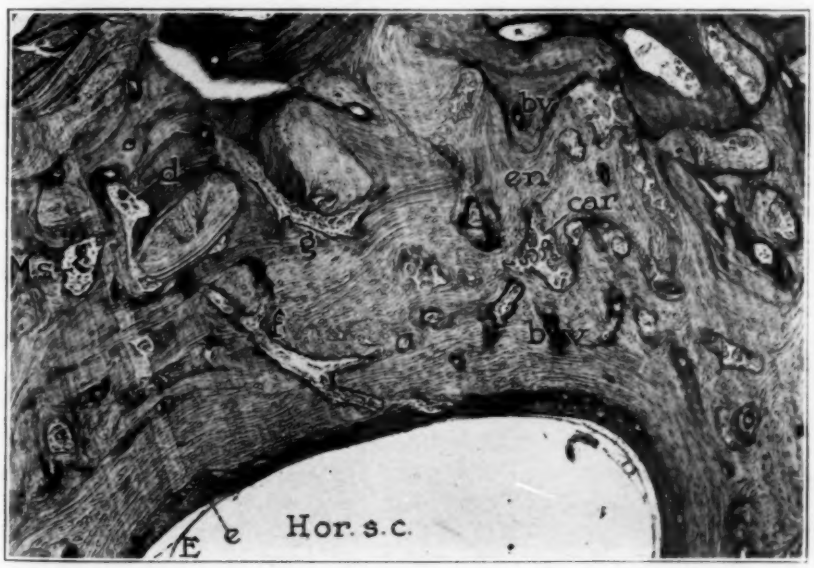

Fig. 8.—Section through the smooth end of the horizontal semicircular canal (*Hor. s.c.*), showing the endosteum (*E*), the endosteal layer (*e*), the bone of the endochondral layer (*en*), normal blood vessel channels (*bv*), cartilaginous interglobular spaces (*car*), marrow spaces with capillaries but without the so-called *Grenzscheide* (*M.S.*), marrow spaces with capillaries and osteoid at the margins (*d*), an enlarged blood vessel channel with Howship's lacunas but without osteoclasts at the margins (*f*) and enlarged blood vessel channels with osteoid material at the margins (*g*).

of the sagittal semicircular canal there were many small marrow spaces situated within the enchondral layer. The margins of these marrow spaces were covered by a thick blue line (*Grenzscheide*) and were entirely aplastic. Some, however, revealed different conditions, in which the marrow spaces contained dilated capillary, and between the wall of this capillary and the margins of the marrow space appeared many cells, most frequently small osteoclasts, which tended to destroy the blue line (*Grenzscheide*). As the destruction progressed, the *Grenzscheide* disappeared entirely and the osteoclasts destroyed the lamellar bone and altered the haversian channel into a haversian space (figs. 8 and 9). Some of these haversian spaces invaded the endosteal layer of the semicircular canals, but they did not perforate into the perilymphatic space. Occasionally, osteoblasts and osteoid bone were found within these spaces, but in general the destruction of bone prevailed over the formation of new bone. It must be emphasized that all

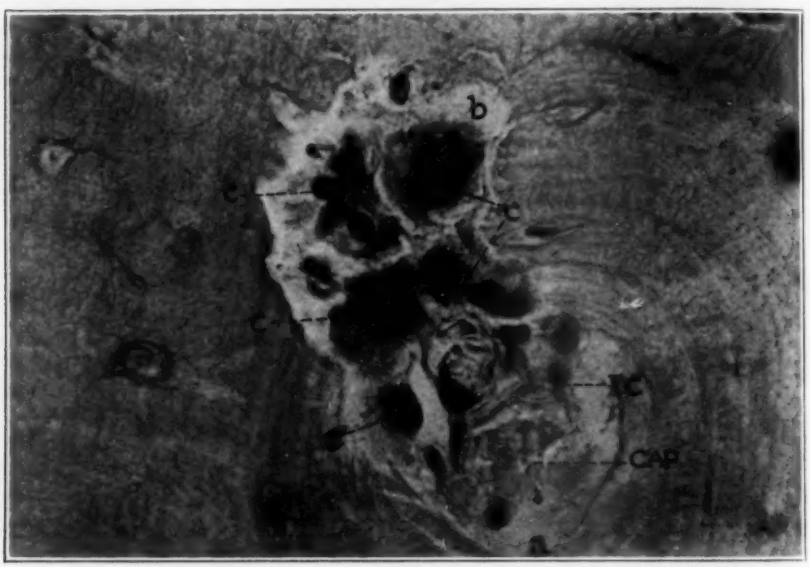

Fig. 9.—Section through a small marrow space in the vicinity of the smooth end of the sagittal semicircular canal on the right side. The inner margin of the old haversian canal (*a*), the newly formed marrow space (*b*) and a large capillary (*CAP*) area seen. Giant cells (*c*) and endothelial cells (*EC*) are also present.

of these changes of the bone were found within the enchondral layer and that none of the newly formed haversian spaces contained pigment.

Comment.—The microscopic changes in this case may be divided into three groups:

1. Congenital deformities.

2. Abnormal destruction of the bony capsule of the semicircular canals.

3. Hyperpigmentation.

To the congenital deformities belong such conditions as the narrowing of the scala tympani of the basilar portion of the cochlea on the left

side, the presence of loose connective tissue in the mesial and inferior angle of the scala tympani of the basilar portion of the cochlea on the left side and the absence of subdivisions in the spiral ganglion of the basilar portion on the left side. All of these findings point to an abnormal course in the development of the inner ear and they support the general opinion that in the presence of a melanoma not infrequently disturbances in the development of different tissues are observed. This holds true particularly if the melanoma develops early after birth, as was the case in the patient whose case has been described here. As far as the slight atrophy of the spiral ganglion and nerve on the left side is concerned, it cannot be stated definitely whether this also is due to a disturbed development of the inner ear or to the tumor which was found close to the left cerebellopontile angle. Inasmuch as this atrophy is not present on the right side, the latter explanation seems more likely to be the true one.

Interesting is the finding of osseous destruction in the bony capsule of the semicircular canal on the right side. From the classic examination of Mayer [14] it is known that after the second year of life there are no destructive changes of the enchondral capsule of the inner ear. Instead, a narrowing of all marrow spaces and blood vessel spaces by the production of new bone through osteoblasts is observed. This patient was 11 years of age, and inasmuch as definite destructive processes were observed in the enchondral capsule, these findings must perforce be called pathologic. The question arises, however, as to what the reasons are for these changes. The first thought would be that these destructive changes point to an abnormal course of the development of the enchondral capsule, in accord with the other congenital deformities mentioned. If such an explanation were correct, one should expect to find these destructive changes on both sides.

Unfortunately, the inner ear on the left side was not completely at my disposal. However, even under these circumstances the explanation pointing to an abnormal course of development cannot be accepted. Inasmuch as the destructive processes were found in only one part of the inner ear, namely, in the area surrounding the smooth end of the sagittal and horizontal semicircular canal, and not in the entire enchondral capsule, and since that area is very close to the posterior fossa, it is obvious that the explanation for these destructive processes has to be sought for in the changes within the posterior fossa itself. As a matter of fact, the melanoma of the dura was found within the posterior fossa, and thus it can be understood readily that the destructive processes were due to the pressure of the tumor in the posterior fossa.

14. Mayer, O.: Die Otosklerose, Leipzig, A. Hölder, 1917.

This explanation, however, encounters one difficulty, because no such destructive processes in the enchondral capsule of the semicircular canals are found when the inner ear is examined in tumors of the cerebellum or of the cerebellopontile angle. However, there is one fact which also obviates this objection. It must be remembered that in the case which has been reported here the tumor apparently developed soon after birth—probably immediately thereafter—consequently, at a time in which the enchondral capsule had not reached its full maturity. Tumors of the cerebellum and pontile angle previously mentioned as a rule develop in a later period of life, consequently, at a time in which the enchondral capsule of the inner ear has already reached its maturity. It is therefore obvious that the tumor in this case must have influenced the enchondral capsule in a different manner than do tumors which develop in a later period of life.

The outstanding finding in this study consists of the hyperpigmentation of the inner ear. That such a hyperpigmentation was present was proved by the macroscopic appearance of the temporal bones, because the walls of the vestibulum, as well as those of the bony cochlea, had a black appearance. However, the microscopic examination furnished further proof that this diagnosis was correct. According to the classic examination of Alexander,[7] the distribution of the pigment within the inner ear is as follows:

1. Pigment is always found: (*a*) in the immediate surroundings of the cristae and macula, where the pigment lies under the layer of epithelium and (*b*) in the vascular stria, where the pigment lies between the connective tissue cells.

2. Pigment is never found: (*a*) within the cristae, the macula and Corti's organ and (*b*) in the epithelial wall of the semicircular canals.

3. Pigment is occasionally found: (*a*) in the bony floor of the vestibulum, where it lies in the small narrow spaces immediately under the surface, (*b*) in the membrana tympani secundaria, (*c*) in the epithelial wall of the sacculus, (*d*) in the mesial part of the utricle and (*e*) in the area of the sinus utricularis superior and posterior.

4. Pigment is rarely found: (*a*) along the membranous walls of the semicircular canals, (*b*) in the intermesial part of the perilymphatic tissue, (*c*) in the endosteal layer or in the wall of the vessels of the bone, (*d*) in the cristae spiralis, lamina spiralis or in the periosteal layer of the scalae, (*e*) in the periosteum of the internal meatus, (*f*) in the connective tissue of the axis of the cochlea, (*g*) in the fossa subarcuata, (*h*) in the mesial wall of the middle ear and (*i*) in the ossicles.

These statements of Alexander concern, of course, the histologic changes in temporal bones only of persons of the white race. As far as I know, the pigment of the inner ear in Negroes has not been studied

specifically or reported until this time. However, granted that there is an increase of pigment in Negroes, in my experience this increase never reaches such a degree that the pigment can be seen macroscopically in the aforementioned sites. Furthermore, when one considers the fact that in lower mammals, which show a varying distribution and intensity of body pigment, the pigment of the inner ear does not vary markedly, it can be assumed that in human beings, likewise, the distribution and intensity of the body pigment will not have a marked effect on the pigment of the inner ear.

If Alexander's concept of the normal distribution of pigment is compared with the distribution found in the case reported here, the following becomes apparent: A definite hyperpigmentation was found in 1*a* but not in 1*b*. The latter point will be discussed later. No pigment was found in 2*a* but was present in 2*b*. A definite increase of pigment was observed in 3*a*, *b* and *d*, while there was no increase in 3*c*. Finally, there was a definite increase in 4*a*, *c*, *d*, partially in 4*f*, and in 4*g*. As far as 4*b* is concerned, nothing can be stated definitely, because the perilymphatic tissue was not preserved in the proper manner. From the foregoing description follows the fact that the microscopic examination also proved a definite hyperpigmentation of the inner ear.

In order to obtain a better perspective concerning the distribution of pigment in the case reported here, I wish to compare this case with one of ochronosis of the temporal bone described by Brunner,[15] in which there was also hyperpigmentation of the ear. In the case reported by Brunner, the distribution of the pigment was almost identical with that in my case. However, there was one important difference. While in the case of ochronosis the peaks of the cristae, the subepithelial layer of the macula utriculi and the cavities for the osteocytes in the periosteal and endosteal layer were pigmented, similar pigmentations were absent in my case. It is difficult to explain this difference. However, one has to bear in mind that there were distinctly different pathologic processes at work in the 2 cases. While in ochronosis, homogentisic acid circulates within the blood and is changed to the ochronotic pigment by the fermentative influence of tyrosinase, in my case, typical melanin was produced by a special metabolic function of the cells, probably by virtue of the influence of oxydasis on products of protein metabolism.

More important, perhaps, is the fact that in neither case did Corti's organ or the stria vascularis evidence an increase of pigment. From this fact one must draw the conclusion that in any kind of hyperpigmentation of the inner ear, the sensory apparatus of the cochlea escapes the tendency to hyperpigmentation. Brunner has explained this fact by pointing to

15. Brunner, H.: Ueber die Veränderungen des Schläfenbeines bei der Ochronose, Monatschr. f. Ohrenh. **63**:997, 1929.

the extremely high development of metabolism within Corti's organ. I do not know whether or not these explanations are correct. However, I should like to draw attention to the observation that in all marrow spaces of the capsule of the inner ear in which a destruction of bone and apposition of newly formed bone was noted, in consequence of which a high degree of metabolism was to be assumed, pigment was absent.

SUMMARY

Histologic examination of the internal ears of a child who succumbed to melanocarcinomatosis of the dura offered an excellent opportunity to study the distribution of pigment in the internal ear. While deposits of pigment in this area have been reported previously, the hyperpigmentation was not so intense as to be observed macroscopically, nor did it occur in association with the pathologic state of melanosis. The findings in this case prove the existence as an entity of melanosis of the inner ear. I do not hesitate to compare this melanosis of the internal ear with melanosis oculi, which is a congenital condition marked by focal or diffuse excessive pigmentation of the choroid, iris or conjunctiva. However, there are two essential differences between these two conditions. First of all, melanosis of the internal ear is less common than melanosis oculi, although slighter degrees of melanosis of the internal ear are met with occasionally. The second great difference lies in the fact that Doherty (after Ewing) [16] found that in 29 per cent of the cases of melanosis oculi melanoma eventually developed, a fact which has led Ewing to call melanosis oculi "the ocular counterpart of the cutaneous nevus." So far as I can determine, a melanoma of the internal ear has never been reported. The study of this case, therefore, afforded an unusual opportunity to study further the biology of melanomas, especially as they concern the internal ear.

Dr. Amil Bach, formerly of the Cook County Hospital, Chicago, furnished the gross specimens.

16. Doherty, cited by Ewing, J.: Neoplastic Diseases, ed. 3, Philadelphia. W. B. Saunders Company, 1928.

LOCAL USE OF SULFANILAMIDE IN OTOGENIC INFECTIONS

FRANK L. BRYANT, M.D.
MINNEAPOLIS

Chemotherapy has become an important means for the effective combating of infections, which previously depended on less specific agents. Sulfanilamide, which was one of the first drugs synthesized, has been used with success against otogenic infections. Many operations for acute mastoiditis have been avoided by early use of this drug.

Investigative studies have shown that under certain in vitro conditions sulfanilamide is effective in its bacteriostatic property in a dilution of 1:10,000. In higher concentrations, however, the bactericidal and bacteriostatic influences may be augmented considerably. Clinically, a blood concentration of free sulfanilamide up to 10 to 20 mg. per hundred cubic centimeters can be obtained. The solubility of sulfanilamide at body temperature is about 0.8 per cent. It has been demonstrated that many substances, including peptone, may interfere with the action of sulfanilamide on micro-organisms.

In an active focus of infection to which the blood supply is impaired, because of either anatomic or pathologic abnormality, sulfanilamide carried by the blood serum cannot be expected to accomplish its purpose.

It has been determined further that sulfanilamide has bacteriostatic and limited bactericidal action in vitro on hemolytic streptococci, staphylococci, pneumococci and colon bacilli. The magnitude of this effect is dependent on the concentration of the drug and on the concentration of the peptone in culture mediums.

Obviously, if a greater and more prompt effect can be obtained by direct implantation, as compared with oral or parenteral administration, disagreeable and dangerous side reactions will be avoided.

Fifteen patients comprised the group for this study. Eleven had chronic otitis media. Three who had undergone radical mastoidectomies had continued purulent exudation from the operative cavity. The first patient treated by this method had acute surgical mastoiditis following an attack of scarlet fever. Preoperative lateral sinus phlebitis was aborted by local application of sulfanilamide.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., June 16, 1941.

ACUTE SURGICAL MASTOIDITIS

A brief résumé of this case is of interest.

J. R. had a severe attack of scarlet fever, the onset of which occurred March 10, 1938. He was placed in isolation, and during the period of isolation immune serum and sulfanilamide were administered. On March 29 otitis media developed on the right side. On the following day spontaneous perforation of the tympanic membrane occurred. The patient's general condition was poor, and he gradually became more toxic. On April 7 the aural discharge became profuse, and roentgenograms taken on this date showed increased density of the right mastoid bone. On April 16 a second roentgenogram was taken. No change in the mastoid was observed. On April 23 roentgenograms revealed definite destruction in the right mastoid process. Clinical findings indicated acute surgical mastoiditis, and on April 25 mastoidectomy was performed. Surgical exposure revealed a large pneumatic cell type mastoid, the walls of which were partially destroyed and contained seropurulent exudate. Culture of material from this area revealed Streptococcus haemolyticus. The sinus plate, which was found to be necrotic in one area, was removed until healthy bone was encountered. In the area directly subjacent to the sinus plate the lateral sinus was covered with dirty gray granulation tissue. A Penrose drain was inserted, and the wound was packed loosely with gauze.

For the first two postoperative days the patient's temperature remained elevated and his condition was very toxic. On the second postoperative day the Penrose drain and the packs were removed and the operative cavity was reinspected. A moderate amount of seropurulent exudate was present throughout the cavity, and the dirty gray granulation tissue covering the lateral sinus appeared more extensive than at the time of operation. As much as possible of the exudate and some of the superficial granulation tissue were removed. Five sulfanilamide tablets were crushed with a hemostat, and the crystals were placed directly over and about the lateral sinus. A Penrose drain was inserted and a light external dressing applied. This procedure of cleansing the operative area and implanting sulfanilamide crystals was carried out daily for seven consecutive days. At the third dressing there was a definite change in the appearance of the operative cavity and especially in that of the granulation tissue. At the end of the seventh postoperative day the entire cavity appeared clean, and healthy granulation tissue was present throughout. The patient's condition was markedly improved. He was discharged on May 10. On May 28 both the operative area and the ear drum had healed completely. The hearing, according to an audiometric test, was essentially normal, and the patient's general condition was excellent.

Comment.—The marked lateral sinus phlebitis found at operation and a more extensive change observed within forty-eight hours indicate that without local sulfanilamide therapy thrombosis of the lateral sinus might have developed. The change in the appearance of the operative cavity after the use of sulfanilamide was so striking that it cannot be overemphasized.

CHRONIC OTITIS MEDIA

During the period 1938 to 1940, 11 patients who complained of "chronic running ear" came under my observation at the University of

Minnesota Health Service, in Minneapolis. Of these, 7 were males and 4 were females. Their ages were from 17 to 28 years. Five of the patients gave scarlet fever as the cause of the aural discharge; 2, measles, and the remaining 4 stated that the otorrhea followed an acute infection of the upper respiratory tract. In 9 of the cases the discharge was so profuse that either cotton had to be worn in the aural canal or the canal and cavity had to be cleansed frequently. In 10 patients tonsillectomy and adenoidectomy had been done with no apparent benefit as far as the aural discharge was concerned. The patient who contracted scarlet fever at the age of 24 had a normal nose and sinuses. A tonsillectomy had been performed in childhood.

In each instance the aural disease was unilateral. The perforations were all central. They varied from ruptures 1 to 2 mm. in diameter to those involving almost the entire tympanic membrane. In 1 patient (M. G.) there was a subtotal loss of the tympanic membrane; the malleus was absent, and granulation tissue almost filled the middle ear cavity.

Roentgenograms were taken of the mastoid area in 7 cases. In 6, "chronic sclerotic mastoiditis" was found. In the seventh (that of L. S.), a normal condition of the mastoid process on the affected side could be demonstrated; the discharge from the affected ear had begun two years previous to this examination, from a nonspecific origin.

In 9 of the 11 cases, local treatment had been given previously, with but temporary or practically no benefit. According to the histories, irrigation with alcohol and treatment with boric acid–alcohol drops or a "powder" had been used singly or in combination. The patients had not been free of otorrhea for any length of time. During previous treatments the discharge had not subsided completely at any time in four years.

In all of the cases smears were made. In each instance a mixed infection was present.

When a patient presented himself for examination and treatment, the middle ear cavity was cleansed carefully by the use of cotton-tipped applicators or by gentle irrigations with 70 per cent alcohol. The aural cavity was dried before any powder was used. A not too heavy film of sulfanilamide powder was insufflated into the middle ear cavity.

The patient was admonished not to get water into the ear or to blow his nose. If he insisted on blowing his nose, he was advised to press the tragus firmly against the orifice of the aural canal.

Five patients who had a history of scarlet fever were given the following number of treatments in order to obtain a dry ear: F. R., two; W. B., one; G. J., two; M. H., five, and P. S., four in one instance and six in a second. Of the 2 patients who had had measles, H. B. required three in the first period and two following an acute attack of nasopharyngitis; D. K. had two.

In 5 of the 11 patients (P. S., F. R., W. B., G. J. and D. K.) the treated ears have remained completely dry. There was neither discharge nor moisture for over one year from the date of the last treatment. Of 4 patients (S. H., H. B., M. H., L. S.) who were seen during 1939-1940, no aural discharge had been present for four months in the first 2 and for five months in the other 2. Examination of the middle ear cavities of all these patients revealed healthy-appearing mucous membranes. In the case of patient M. G. there was roentgen evidence suggestive of an abscess or a cholesteatoma. The discharge had lessened but had not ceased after twenty treatments. Radical mastoidectomy has been advised. One patient (P. S.) showed at each examination a variable amount of thin mucoid secretion instead of the profuse purulent discharge which had occurred previously.

It is significant that in the patients whose middle ear cavities became dry and remained so during an acute infection of the upper respiratory tract, no exacerbation of the otitis media occurred. One might theorize that the insufflated powder not only acted as a barrier to the introduction of infective material into the middle ear via the eustachian tube but also rendered inert any bacteria which were introduced. The sulfanilamide powder was not absorbed, for at subsequent examinations it could be seen macroscopically in the middle ear cavity.

Comment.—The patients were enthusiastic about this type of treatment because of the promptness with which relief was obtained as compared with their previous experiences and the freedom from pain with the insufflations of the sulfanilamide powder as contrasted with alcoholic irrigations or certain insufflations of iodine powder.

CHRONIC OTITIS MEDIA; POSTOPERATIVE RADICAL MASTOIDECTOMY

R. T. stated that trouble with his right ear began when he was 3 years of age, following an acute infection of the upper respiratory tract. Otorrhea was present for thirteen years. Dry wipe treatments and drops were used at various times. The aural condition gradually became worse. He consulted an otologist, who advised him to undergo mastoidectomy. This operation was performed in January 1937. The ear became dry and remained so for two months following the operation. In April the patient went swimming, and on the following day purulent discharge occurred in the right ear. He had treatments for his ear but nevertheless continued to swim at intervals. The ear continued to discharge throughout the summer. In the fall of 1937 local treatment was carried on by an otologist but with no improvement. In the fall of 1938 the patient presented himself for treatment at the health service. The history revealed that the aural discharge had been present since he went swimming in April 1937. Examination revealed a middle ear and operative cavity filled with thick mucopurulent exudate. After the area was cleansed, further examination revealed the scar of a well performed radical mastoidectomy. Smears showed mixed organisms. The area of the antrum and mastoid contained some granulation tissue, and the entire mucous membrane was intensely inflamed. Sulfanilamide powder was insufflated on alternate days until

five treatments had been given. On the sixth visit the aural cavity was essentially dry. The mastoid and middle ear cavity have remained dry with two exceptions. The first exception occurred when the patient got water into his ear during a shower bath. Two treatments, which were given on alternate days, completely checked this exacerbation. A second flare-up occurred immediately after an acute infection of the upper respiratory tract. Two treatments, likewise, cleared up this condition.

Comment.—The infection, probably confined to the mucous membrane and the modified lining of the aural cavities, went deep enough to resist the usual methods of treatment. The insufflation of sulfanilamide apparently produced enough inhibitory or germicidal action to effect a cure.

T. P., aged 26, gave a history of an acute infection of the upper respiratory tract in childhood, followed by an aural infection. A simple mastoidectomy was done, but aural discharge continued for several years. A radical mastoidectomy was performed, but otorrhea, purulent in character, continued, although less profuse. Two years later a revision of the radical mastoidectomy was made. This was followed by local treatment. The purulent discharge, however, continued. The patient was seen first in March 1940. After a moderate amount of purulent exudate was removed, examination revealed the cavity of a radical mastoidectomy. Boric acid drops, boric acid–iodine powder and various germicidal tinctures were used without avail. Sulfanilamide powder was subsequently insufflated, with almost complete cessation of the discharge. Examination revealed granulation tissue in the region of the roof of the mastoid antrum. This tissue was removed. Three additional treatments resulted in drying of the ear. Three months have elapsed since the last treatment. There is no trace of any exudate. The result seems to the patient almost unbelievable.

M. L., aged 30, gave a history practically parallel to that of T. P., with this exception. No granulation tissue had to be removed. Four treatments, on alternate days, produced a dry middle ear and mastoidectomy cavity.

SUMMARY

Pertinent facts concerning sulfanilamide are reviewed. A report is made on 15 patients who were treated for otogenic infections by local applications of sulfanilamide powder: (*a*) 1 with acute surgical mastoiditis and, in addition, lateral sinus phlebitis; (*b*) 11 with chronic otitis media, and (*c*) 3 with purulent otorrhea following a radical mastoidectomy. Sulfanilamide powder proved efficacious in 14 of the 15 patients treated.

CONCLUSION

The local use of sulfanilamide powder is a safe and effective method of treatment for certain otogenic infections.

Since these observations were made with sulfanilamide powder, similar results have been obtained with the use of sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) powder.

Case Reports

BILHARZIASIS OF THE LARYNX

HASSAN BEY SHAHEEN, M.CH., CAIRO, EGYPT

The question of bilharziasis in Egypt is of vital importance to the nation. The disease is sapping the energy of the people, and widespread campaigns are now being conducted with energy in the hope of completely eradicating the disease.

Fig. 1.—Section of tissue from the larynx, showing bilharzic ova in situ. The absence of histologic evidence of local reaction to their presence is noteworthy.

In 1928 Prof. Mohamed Sobhy Bey [1] published a report of a case of bilharziasis of the conjunctiva, the first of its kind.

For several years I have been unsuccessfully looking for a report of bilharziasis of the nose, pharynx or larynx. I have not found any such

1. Sobhy Bey, M.: La bilharziose palpébro-conjonctivale, Ann. d'ocul. **165**: 675, 1928.

condition reported. Prof. Khalil Bey, an eminent parasitologist, told me that bilharziasis in the nose of bovines has been reported [2] but that he did not know of any occurrence of this condition in man. I encountered the disease in a patient with laryngeal obstruction. I believe this is the first case of laryngeal bilharziasis to be reported.

A man aged 35 was admitted to my service in the ear and throat department of the Kasr el Ainy Hospital with the following history:

Two years previously the patient noticed a change in his voice. This was accompanied by difficulty in breathing and by dysphagia. These symptoms increased, and the dyspnea became so severe that tracheotomy had to be done. There was no history of syphilis, and the Wassermann test was negative. Examination for tuberculosis also gave negative results, and a roentgenogram of the lungs showed no abnormality.

Indirect laryngoscopic examination showed the presence of a fungating mass of cauliflower form occupying the area above the vocal cords and hiding them from view. At the angle of the jaw movable nodes were felt.

Tracheotomy was performed, to relieve respiratory distress, and a specimen of the growth was removed for biopsy.

Prof. Soroor Bey reported as follows: "The material submitted consists of vascular fibrous tissue, in which are deposits of fresh and semicalcified bilharzic ova, with no evidence of reaction. The fibrous tissue is covered with squamous epithelium."

This report was confirmed by another biopsy, which showed the same conditions.

As this, so far as I know, is the only case of its kind, it seems worthy of publication.

The photomicrograph of a section of the lesion in this case shows the ova clearly.

Prof. Soroor Bey supplied the photomicrograph.

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2. Indian J. Vet. Sc. & Husbandry, December 1932, vol. 2, pt. 5.

BENIGN ENCAPSULATED FIBROMA OF THE LARYNX

D. F. Weaver, M.D., and J. L. Dill, M.D., Detroit

B. A., a white man aged 31 years, presented himself at the Henry Ford Hospital on Oct. 14, 1939. He stated that eight years before admission hoarseness had begun and that it had increased gradually to such a point that he could speak only in a loud whisper. Recently, there had been moderate respiratory obstruction, which was increased when he smoked cigarets. Five and one-half years ago he had undergone an operation on his larynx, in Europe. This did not improve the condition. A short time afterward he consulted a laryngologist who advised cauterization of his vocal cord, but since the laryngologist gave him no assurance as to how much voice improvement would be obtained he had nothing done and did not seek medical attention again until his admission to Henry Ford Hospital. There was no other history of disease of the ears, nose or throat.

On examination of the ears, nose and throat, a moderate amount of pus was found in the right superior nasal meatus. The tonsils were chronically infected,

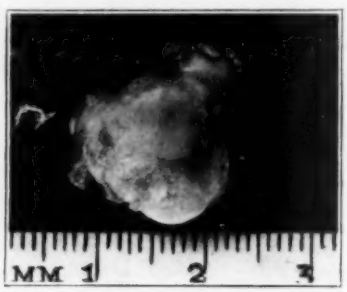

Fig. 1.—Photograph of the removed fibroma.

but this was thought not to be clinically significant. Examination of the larynx revealed a tumor, which appeared to be about 1 cm. in diameter, involving the anterior half of the left vocal cord and located in the substance of the vocal cord, pushing it medially rather than attached to it. It appeared to be round, firm and benign. The tumor could not be palpated in the neck. Neither the remainder of the examination of the ears, nose and throat nor the general examination revealed any abnormality.

In view of the finding of pus in the right side of the nose, the patient was sent for roentgenograms of the sinuses. These showed maxillary sinusitis on the right side. The right antrum was washed out for diagnosis, and a large amount of pus was obtained. As it was believed that he would have general anesthesia for removal of the laryngeal tumor, it was thought advisable to clear up the infection in the antrum first. Consequently, on October 20 a Caldwell-Luc operation was carried out on the right antrum. The mucous membrane lining the antrum was found to be swollen and polypoid, and a moderate amount of pus was noted. A large window was made in the nasoantral wall. The nasal condition cleared up without incident, and on November 17, approximately a month

after the nasal operation, with the patient under anesthesia induced with ether and nitrous oxide, the larynx was exposed by the use of the Lynch suspension laryngoscope.

We had hoped to make a biopsy of this tumor and, if possible, to remove the growth piece by piece, since it was believed to be probably a soft tumor. An

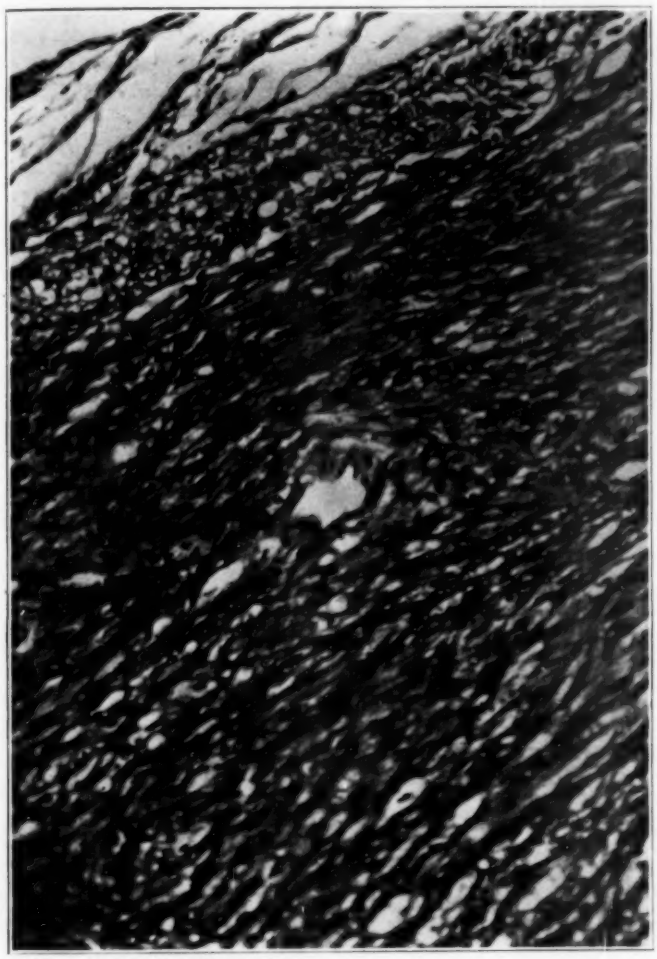

Fig. 2.—Photomicrograph of the fibroma.

attempt to take a specimen for biopsy, however, resulted only in removal of that portion of the mucous membrane which covered the tumor. After this membrane was removed, attempts to get a specimen from the tumor itself were unsuccessful, because of the extreme hardness of the growth, and resulted in slipping off of the instrument each time. It was then decided that an attempt should be made to dissect the tumor out in its entirety. This attempt was successful, and the tumor was dissected out in much the same way as a tonsillar

sebaceous cyst or any benign tumor. A tracheotomy set was kept at hand in case of an emergency. The patient was sent to his room in good condition. The tumor was photographed and then sent to the pathologic laboratory, where a diagnosis of benign fibroma was made. The specimen measured 2.2 by 1.6 by 1 cm. Several days after the operation the patient had some voice, and after several weeks the voice was quite satisfactory and has remained so.

It is frequently stated that fibroma and papilloma constitute a large majority of the benign tumors removed from the larynx. That statement is true when inflammatory tumors that have undergone fibrosis are included. These tumors, however, are never circumscribed and encapsulated. True encapsulated fibroma is rare. New and Erich [1] reported 722 consecutive benign tumors of the larynx which were removed over a period of thirty years. In this group there were 6 diagnosed as true fibroma, an incidence of 0.8 of 1 per cent. All 6 tumors were soft. They measured from 2.5 mm. to 2.5 cm. in diameter. No hard encapsulated fibroma within the substance of the vocal cord was reported in this series.

No such tumor has been encountered previously at the Henry Ford Hospital.[2]

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1. New, G. B., and Erich, J. B.: Benign Tumors of the Larynx, Arch. Otolaryng. **28**:841-910 (Dec.) 1938.

2. Hartman, F. W.: Personal communication to the authors.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

CHRONIC PROGRESSIVE DEAFNESS, INCLUDING OTOSCLEROSIS AND DISEASES OF THE INNER EAR

GEORGE E. SHAMBAUGH JR., M.D.
AND
WYATT S. ROBERTS, M.D.
CHICAGO

The literature for 1940 reflects the war in the paucity of worth while contributions from the belligerent countries, in a valuable article on functional deafness in soldiers and in one on aviation deafness. The surgical treatment of otosclerosis and the relation of vitamins to deafness continue to share the spotlight of interest, if one can judge by the number of articles on these two subjects. As in previous reviews, my editorial comments are in brackets. The material is arranged as follows:

Otosclerosis
- Pathology and Etiology
- Treatment

Chronic Adhesive Deafness

Labyrinthine Deafness
- Traumatic Nerve Deafness
- Nerve Deafness from Infection
- Nerve Deafness from Endocrine Disturbances
- Nerve Deafness from Avitaminosis
- Treatment of Nerve Deafness
- Miscellaneous Aspects of Nerve Deafness
- Ménière's Syndrome

OTOSCLEROSIS

Pathology and Etiology.—Covell [1] studied 46 temporal bones from persons with otosclerosis and 21 bones from persons without otosclerosis, as controls, directing his attention to any changes in the ossicles. In both ears of one otosclerotic patient a typical otosclerotic focus was found in

1. Covell, W. P.: The Ossicles in Otosclerosis, Acta oto-laryng. **28**:263, 1940.

the head of the malleus and in the incus. This is the first time that a focus of otosclerosis has been reported outside the labyrinthine capsule.

In the otosclerotic bones with extensive ankylosis of the stapes there was observed to be marked displacement of the malleoincudal or incudostapedial joints, and when the stapes was not ankylosed but the oval window was distorted by the otosclerotic focus, there was moderate displacement of these joints. The author attributes these displacements to the fixation of the stapes with abnormal movements of the remainder of the ossicular chain and attempts on the part of the ossicles to overcome the resistance in the stapediovestibular joint. Cartilaginous rests and interglobular spaces in the ossicles indicate a preotosclerotic condition similar to that found in the labyrinthine capsule.

Bast,[2] continuing his fundamental studies of the otic capsule, states that the occurrence of residual cartilage or defective ossification in certain parts of the petrous bone in late fetal life or in early childhood has been recognized for some time. This defective development occurs in the region of the fissula ante fenestram in a large percentage of fetuses and young children. It has been suggested by this author and others that otosclerosis is a late attempt at rebuilding of bone in this unstable region.

Since otosclerotic bone occasionally occurs elsewhere in the labyrinthine capsule, the author examined 64 older fetal and 28 infant petrous bones, to determine whether residual cartilage is found in these other areas. Residual cartilage was found in four locations:

1. In the posteroinferior portion of the bony capsule of the cochlea below the internal auditory meatus. This cartilage mass usually disappears at about the time of birth, but the bone formed from it is much looser and poorer in type than the rest of the capsule and may therefore be subject to reorganization and perhaps to an otosclerotic process.

2. The styloid bone and adjacent bony process develop from cartilage which remains cartilaginous at the base of the process until after birth. Since the styloid process is rarely included in sections of the petrous bone, otosclerotic changes usually are not noted.

3. In the region of the petrosquamous suture, anterior to the posterior and horizontal semicircular canal and lateral to the facial nerve, a cartilaginous suture was found in 50 per cent of the infant bones examined. In a certain number of cases ossification of this cartilage appears to be faulty. There are no known cases of otosclerosis in this region.

4. Isolated spots of cartilage persist in the bony capsule of the canal region in about 65 per cent of petrous bones representing the late fetal

2. Bast, T. H.: Development of the Otic Capsule, Arch. Otolaryng. **32**:771 (Oct.) 1940.

period and the period of infancy and childhood up to 3 years of age. However, since they lie outside the endochondral bone, while otosclerotic foci have been found in the endochondral bone in this region, there is no apparent relationship between the two. There is one exception to this—a case reported by Siebenmann, in which the location of the otosclerotic focus corresponded to that of residual cartilage.

[Comment: Bast adds more evidence in favor of his theory that otosclerosis represents a late attempt at rebuilding of bone in unstable regions of the labyrinthine capsule. His theory appears to be the most acceptable thus far advanced for the pathogenesis of otosclerosis.]

Slaughter [3] studied the temporal bones of guinea pigs, rabbits and dogs to which he had previously administered dihydrotachysterol while they were kept on a diet poor in calcium and free from vitamin D. The majority of these temporal bones showed "a sharply circumscribed formation of new bone in the labyrinthine capsule," but the conditions found showed great variation. This new bone could be differentiated from the normal osseous tissue by its deep-staining characteristics, by the marked enlargement of the bone spaces and by the haversian canals. The pathologic bone was lacking in detail, the normal mosaic being absent. In certain areas marked bony metabolism or formation was evidenced by the presence of numerous osteoblasts or osteoclasts, without any line of demarcation. The reaction closely resembled that of callus formation following fracture, in which marked bony metabolism is seen. Bony ankylosis of the stapes footplate was not observed, but the author suggests that it might have been had the experiment been carried further. The author concludes that hyperparathyroidism is one of the causes of otosclerosis and that by the administration of dihydrotachysterol, which he calls a parathyroid hormone, he had produced the lesion of otosclerosis.

[Comment: Since otosclerosis is characterized by its local occurrence in the temporal bone without demonstrable changes elsewhere in the skeleton, whereas in hyperparathyroidism the bony changes occur throughout the skeleton, and since otosclerosis has never been described in clinical hyperparathyroidism, the author's conclusions seem entirely unjustified. Likewise, his statement that dihydrotachysterol is a parathyroid hormone is open to question.]

Anderson and Nylén [4] studied the temporal bones of mice which had received large doses of fish liver oil or pure ergosterol. Degenerative

3. Slaughter, E. C.: Experimental Hyperparathyroidism and Otosclerosis, Ann. Otol., Rhin. & Laryng. **49**:130 (March) 1940.

4. Anderson, H. J., and Nylén, C. O.: Histologic Study of Temporal Bone of Mice with Hypervitaminosis and of Pregnant Mice, Acta oto-laryng. **28**:176, 1940.

foci were found which resembled the foci of otosclerosis observed in man. In pregnant mice which had received the same medication these foci resembling otosclerosis were not seen, but rather a well defined osteoporosis. The authors conclude that they cannot be sure that the changes observed have anything in common with human otosclerosis. Their observations suggest that hypervitaminosis may be more of a factor in the pathogenesis of otosclerosis than hypovitaminosis.

Engström [5] studied the temporal bones from 100 random autopsies, those of 50 men and 50 women from 20 to 90 years of age, a total of 145 temporal bones being examined. In 12 specimens otosclerotic changes were observed; 8 of the bones were of the typical spongy type, while in the remaining 4 the so-called blue mantels were present. In 6 cases in which both ears were examined, the changes were bilateral and rather symmetric. In most cases the otosclerotic changes represented the rather early stage of development of the disease, and in no case was there bony ankylosis of the stapes footplate, but in 5 the changes lay within the typical region in front of the oval window, with fibrous thickening of the ligament of the stapes footplate. In 2 cases there were well localized otosclerotic changes within the bony capsule of the labyrinth, without connection with the regions which are significant for sound conduction or perception.

Many transitional stages between typical spongy otosclerotic changes and the so-called blue mantels are evidence that the latter represent a special stage of development in the course of otosclerosis. Several observations of compression of the oval window and deformity of it are evidence that mechanical factors may be significant for the development of otosclerotic changes. The author has been able to prove experimentally that injuries of the bony labyrinth can lead to local rebuilding processes within the uninjured bone.

All 12 of the otosclerotic temporal bones were from persons above 40 years of age. Of the temporal bones from 30 persons between 20 and 40 years of age none were otosclerotic. Of the 12 patients with otosclerosis 8 were men, 4 were women.

Engström then reports the histologic findings in experimental labyrinthine fistulas made by Holmgren and his associates in monkeys. Their experiments were done to show the normal healing processes in such a fistula and to show how different substances used to cover the fistula influence these healing processes.

The author found that when a Stacke chisel is used to make the fistula, connective tissue closure begins within a few days and that after

5. Engström, H.: Ueber des Vorkommen der Otosklerose, nebst Experimental über chirurgische Behandlung der Krankheit, Acta oto-laryng., 1940, supp. 43.

about one week bone formation begins in the form of irregular callus plugs. After twenty-five to thirty days the fistula has been closed by newly formed connective tissue, osteoid and bone.

If the fistula is made with a rapidly rotating polishing burr, the formation of new bone seems to be checked to a certain degree, as previously demonstrated by Canfield. Unpublished studies by Engström and Orell show that this effect seems to be largely the result of heat; with a bûrr cooled by carbonic acid the formation of new bone does not seem to be impeded to an equal extent.

The size of the fistula has a determining influence on the type of closure. If the circumference of the fistula is less than half that of the canal, the closed canal will be somewhat smaller but rather spherical. If the fistula includes more than half the circumference of the canal, then the entire canal will become obliterated with new bone and the membranous canal will remain as a small tube in the new-formed bone, so that the endolymphatic tissue resists injury. It is therefore of the greatest importance not to make too wide a fistula.

The location of the fistula is likewise important, and the author advises against a fistula too close to the ampullar portion of the canal, because of the risk that new bone will grow into the ampulla, destroying the sensory epithelium and possibly preventing a second operation on the same canal.

The new bone around the fistula is at first composed of irregularly arranged fibrils, which are gradually transformed into lamellar bone. This transformation proceeds very slowly, and only in isolated cases can one observe osteoclastic resorption.

Bone splinters left in the fistula seem to exert a stimulating effect on new bone formation. These bone splinters remain unchanged within the new-formed bone as long as four hundred and sixty-five days after the operation. The complete removal of all bone splinters in the region of the fistula is therefore extremely important.

When the fistula is covered with mesodermal tissue, such as fat, connective tissue or muscle, the closure of the fistula seems to be more rapid, as though this mesodermal tissue constitutes a good foundation for the new-formed bone tissue.

Paraffin, hard rubber and lens of the eye laid against the fistula do not seem to impede the closure to any noticeable degree.

Thiersch grafts and patch grafts from the tympanic membrane have a definite inhibiting effect on bone formation, but this is not sufficient to prevent closure of the fistula.

Thin sheets of metal placed against the fistula impede the formation of new bone to a certain extent. The result varies with the metal used, copper having a great impeding effect, silver less and gold more. The effect seems to be both chemical and mechanical.

A cylinder of stainless steel containing radium placed against the fistula produces a considerable impedance of new bone formation, but this effect seems to disappear in rather a short time after the removal of the radium.

Finally, attempts to maintain an open fistula by placing a small laminaria peg in the fistula were unsuccessful, these pegs seeming to exert a marked stimulating effect on the formation of both connective tissue and bone.

[Comment: Engström's excellent article constitutes the first detailed and extensive work on experimental labyrinthine fistulas in animals. The failure of Holmgren and his associates to secure a permanently open fistula in monkeys is in agreement with their clinical experience with this same technic—all the fistulas closed. These experiments should now be repeated using a technic which clinically has succeeded in keeping the majority of fistulas open.]

Geshelin [6] sought to determine the relation between the endocrine glands and otosclerosis by examining the function of these glands in 35 cases of pure otosclerosis. A polyglandular dysfunction was found to be present, involving chiefly the thyroid and sex glands. For the most part there was hypofunction, but in a few cases there was hyperthyroidism. These endocrine disturbances probably could activate a latent tendency to impairment of hearing. The author regards otosclerosis as part of a general weakness of the mesoderm, which is also manifested in blue scleras and in weakness of connective tissue and of the walls of the blood vessels. He applies the term "neuro-vaso-organo weakness" to pure otosclerosis. He does not advise the feeding of a mixture of endocrine glands in this disease.

Zorbach,[7] as the result of an examination of the constitution of 21 otosclerotic patients, verifies the theory that otosclerosis is based on an inferiority of the mesenchyma. A prevalence of hyperplastic asthenic persons with a weakness in their connective tissue was evident. This, he believes, is further proof of a hereditary basis for otosclerosis.

Fox and Sweet [8] reported a brother and a sister with brittle bones, blue scleras and deafness. Their father suffered from the same combination of symptoms, and three children of the brother, ages 4, 6 and 9, have blue scleras and brittle bones but as yet have no deafness. The brother's loss of hearing began at the age of 30, was progressive and

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6. Geshelin, A. I.: Pathogenetic Structure of Otosclerosis, Zhur. ush., nos. i gorl. bolez. **16**:20, 1939.

7. Zorbach, H.: Constitution of Otosclerosis Patients, Inaug. Dissert., Frankfurt, 1939.

8. Fox, M. S., and Sweet, S. J.: Brittle Bones Associated with Deafness and Blue Scleras, Arch. Otolaryng. **32**:506 (Sept.) 1940.

involved an impairment of conduction with considerable nerve changes in the presence of normal drum membranes and eustachian tubes. The sister's hearing loss began at the age of 24, was progressive and consisted of an impairment of conduction with little nerve change in the presence of normal drum membranes and eustachian tubes. The author directs attention to previous histologic work which showed that the deafness in this syndrome is due to typical otosclerotic foci in the labyrinthine capsule.

Of theoretic interest in regard to chronic progressive deafness is an article by Kobrak, Lindsay and Perlman.[9] Utilizing the reflex contractions of the tensor tympani and stapedius muscles in response to sound the authors led sound through a small tube directly to the round window of a cat and demonstrated that cochlear stimulation by this route is surprisingly effective. The authors name this "reversed sound conduction in the inner ear," since the vibrations now enter the scala tympani rather than the scala vestibuli, as normally occurs.

After destruction of the stapes, hearing by bone conduction was still present, indicating that normal bone conduction is not necessarily via the stapes.

Occlusion of the oval window with plaster of paris resulted in a slight increase of hearing by bone conduction. When both windows were occluded by plaster of paris, there was no hearing by either air or bone conduction, proving that fluid waves in the intralabyrinthine fluid are essential for hearing.

Pressure changes in the cranial cavity are communicated easily to the labyrinth, resulting in similar changes in the labyrinthine fluid pressure. This indicates that glaucoma of the labyrinth due to increased perilymph pressure cannot occur as long as the normal communications with the cranial cavity are open.

[Comment: The observation that occlusion of both windows results in complete loss of all hearing seems to invalidate the round window graft operation for all cases of deafness due to occlusion of the oval window, whether from otosclerosis or chronic adhesive processes. The fact that none of Hughson's patients have experienced total loss of hearing is probably due to the fact that the graft never completely occludes the round window niche.]

Treatment.—Hughson[10] discusses the indications for his round window graft operation for deafness. The operation is not suitable for patients with hearing losses below the general level of 50 decibels. It

9. Kobrak, H. G.; Lindsay, J. R., and Perlman, A. B.: The Next Step in Auditory Research, Arch. Otolaryng. **31**:467 (March) 1940.

10. Hughson, W. A.: Summary of Round Window Graft Operations Performed for Deafness, Ann. Otol., Rhin. & Laryng. **49**:384 (Jan.) 1940.

should not be performed on patients over 50 years of age, but a moderate degree of nerve deafness is not a contraindication. The author also reviews the experimental basis for his operation. While the physiology of the ear was being investigated by the Wever-Bray technic, it was noted that cotton in the niche of the round window increased the intensity of the response. Tissue grafts in the round window also resulted in an increased response as compared with that in the ear which was not operated on. The explanation is apparently that the round window membrane acts as a safety valve to sounds of high intensity. By fixation of the membrane all sounds are heard with a greater intensity.

[Comment: The improvement in hearing observed by the author on occluding the round window has not been observed by other investigators. The observations made by Kobrak, Lindsay and Perlman [9] indicate that closure of the round window should result in a further loss of hearing in cases of conduction deafness.]

Hughson and Witting [11] state that in view of the expected range of variation no change in the hearing level of any frequency less than 10 decibels can be considered a matter of the slightest consequence. Because the thresholds of hearing for individual frequencies do not represent the actual hearing level for speech intelligibility, the authors believe that the routine employment of some type of intelligibility test seems desirable. Finally, because the five year cure has become more or less a standard for determining a satisfactory end result in the majority of pathologic conditions, the authors express the belief that a patient should have the assurance of maintaining any gain obtained for at least this length of time.

[Comment: While the audiogram does not represent the exact hearing level for speech, to date there is no more satisfactory method of recording hearing acuity and hearing improvement after therapy. The "five year cure" has grown up as the standard in the treatment of malignant tumors. There is no particular reason for using it as the standard of hearing improvement. As a matter of experience I have found that a hearing improvement of more than six months after the fenestration operation may be regarded as a lasting improvement—rarely will there be an appreciable loss after this period. Probably the "one year cure" will be found to be entirely acceptable for the end results of therapy for deafness.]

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11. Hughson, W., and Witting, E. G.: Estimation of Improvement in Hearing Following Therapy of Deafness, Ann. Otol., Rhin. & Laryng. **48**:368 (June) 1940.

Hughson [12] presents his results with his round window graft operation in a series of 30 patients operated on over a three year period, the earliest being operated on forty-one months before his article was written, the latest, six months before. After reiterating that "no audiometric improvement of the threshold of hearing representing less than a sustained level of 10 decibels can be regarded as of any real consequence," the author presents a table containing the complete summarized data on his cases. The preoperative hearing level was established by averaging three preoperative audiograms, and the result was estimated by averaging the three most recent audiograms. When the three speech frequencies are averaged and the final postoperative level is compared with the preoperative level, the decibel gain or loss in hearing for speech is as follows:

Case 1.....	Right ear	+9	Case 12.....	Right ear	+1	Case 23.....	Right ear	+4
	Left ear	−9		Left ear	−3	Case 24.....	Left ear	−2
Case 3.....	Left ear	−3	Case 14.....	Left ear	+5	Case 25.....	Right ear	−4
Case 4.....	Left ear	−2	Case 15.....	Right ear	+9	Case 26.....	Right ear	+7
	Right ear	+9	Case 16.....	Left ear	−3		Left ear	+3
Case 6.....	Right ear	0	Case 17.....	Right ear	+1	Case 27.....	Right ear	+10
Case 7.....	Right ear	−3	Case 18.....	Right ear	+6	Case 28.....	Right ear	−2
Case 8.....	Right ear	−6	Case 19.....	Left ear	+9	Case 29.....	Left ear	+2
Case 9.....	Right ear	−2	Case 20.....	Right ear	+2	Case 30.....	Left ear	+5
Case 10.....	Right ear	0	Case 21.....	Right ear	+5	Case 31.....	Right ear	+6
Case 11.....	Right ear	0		Left ear	+5	Case 32.....	Right ear	+3
	Left ear	−10	Case 22.....	Left ear	+6	Case 33.....	Right ear	+8

The author then presents the average decibel gain for each frequency for each month after operation, and in another table the 20 cases with the most favorable results are summarized. These show an average gain for the critical frequencies (512, 1024 and 2048) of 7.8 decibels nineteen months after operation.

The author concludes that his statistical analysis indicates a trend toward improvement in the patients on whom he operated. Subjective improvement was experienced by 60 per cent of all the patients and by 85 per cent of those with favorable results. In 5 out of 15 patients tinnitus was alleviated and in 1 patient it was worse. In no patient was the hearing impaired as a result of this surgical procedure.

[Comment: Hughson's table presenting the complete data in each case and his method of estimating the actual hearing result by averaging the results for the critical frequencies from three preoperative and the three latest postoperative audiograms deserve commendation and together should serve as a model for others reporting the results of therapy for deafness. The only imperfection in his report is the failure to state the hearing in the ear which was not operated on. This provides the best control in any surgical treatment for deafness. One wonders whether the ears on which operation was not performed did not show the same slight "trend toward improvement" as those which were

12. Hughson, W.: An Appraisal of Four Years' Experience with Round Window Grafts for Deafness, Arch. Otolaryng. **32**:611 (Oct.) 1940.

operated on. From Hughson's admirable report and from his own statement that no improvement of less than 10 decibels can be regarded as of any real consequence, the round window graft operation for deafness seems to be of negligible value. Were other treatments for deafness to be reported in a similarly thorough and critical manner, otologists would have a much easier time evaluating these treatments. Because of the increasing interest in the treatment of deafness, it might be a useful measure for the American Otological Society or the editorial boards of the leading journals to require any article on the therapy of deafness to measure up to the standards set so ably by Hughson.]

Goodyear [13] suggests a simplification of the Lempert fenestration technic, in which the conventional postauricular approach is used, the bony meatal wall is taken down to the level of the annulus tympanicus, but without removing the sulcus tympanicus, and a skin flap is cut from the posterior wall of the external auditory canal and turned back to cover the horizontal semicircular canal. The fistula in the semicircular canal is made with a sharp Richards curet. The external auditory meatus is enlarged by a circular flap, which is turned up and back and sutured to the periosteum. The operation has been used on a patient who shows a positive reaction to the fistula test three and one-half months after operation, but no mention is made concerning the hearing.

[Comment: Goodyear's operation is almost identical with the Heath modified radical mastoidectomy. The important question of whether or not the hearing can be substantially improved without removing the attachment of the annulus above and without removing the head of the malleus has not been answered by the author. The use of the postauricular incision, necessitating a later plastic procedure on the meatus, seems an unnecessary and time-consuming addition to the simple and satisfactory endaural incision and approach.]

Porta,[14] using the postaural approach, makes a fistula into the horizontal semicircular canal, exposing the membranous canal as widely as possible and covering the fistula with a part of the aponeurosis of the temporal muscle. An opening into the external auditory meatus to this aponeurosis is then made. The middle ear is not opened. The improvement in hearing is often only transient, because the fistula in the labyrinth closes frequently.

[Comment: Engström [5] has shown by experiments on monkeys that mesodermal tissue such as the aponeurosis of the temporal muscle

13. Goodyear, H. M.: Post-Auricular Approach in Operative Treatment, Arch. Otolaryng. **31**:451 (March) 1940.

14. Porta, C. F.: Contribution to the Surgical Treatment of Otosclerosis, Atti R. Accad. d. fisiocrit. di Sina **8**:215, 1940.

stimulates bony closure of the fistula and is therefore unsuitable for covering the fistula.]

Nasiell [15] believes that it is not possible to separate otosclerosis from chronic adhesive deafness, an artificial permanent fistula being of equal value in both diseases. In this author's opinion, the problem of a permanent fistula is not solved yet. To explain the improvement sometimes observed in the ear which has not been operated on, in 2 cases the mastoid was chiseled against with a blunt instrument. In 1 of these there was an improvement lasting two days, suggesting that vibrations of the skull might account for the improvement in the ear which was not operated on. Suboccipital puncture with drainage of cerebrospinal fluid, by the method of M. Meyer, was carried out in 7 cases. No improvement in hearing was achieved.

[Comment: Because chronic adhesive deafness is usually the end result of suppurative otitis media, after which the hearing defect remains stationary, in my opinion the history generally permits a satisfactory differentiation of this condition from otosclerosis. In my experience with more than 100 cases of otosclerosis in which operation was performed by the fenestration technic of Lempert with certain modifications, the ear not operated on has not shown any significant improvement. Any apparent improvement has been within the limits of normal variation or can be explained as improvement with practice.]

Holmgren [16] presents 4 cases of otosclerosis in which operation was performed by various technics. In case 1 a conservative radical mastoidectomy was followed after eleven months by the formation of a fistula 3 mm. long in the posterior vertical canal. Radium (1 mg.) was inserted over the fistula. Four months later hearing tests showed a gain of 15 decibels for the frequency of 512, a gain of 5 decibels for 1024 and a gain of 20 decibels for 2048, an average gain for these conversational frequencies of 13 decibels. In case 2 a one stage operation was carried out, a fistula 2 mm. long in the posterior vertical canal being covered with cellophane. The cavity was filled with paraffin and 0.24 mg. of radium, which was left in for two weeks. Nine months later the hearing for the tone 512 showed a gain of 25 decibels, that for 1024 a gain of 25 decibels and that for 2048 a gain of 37 decibels, an average gain for these speech frequencies of 29 decibels. In case 3, in a one stage operation a 3 mm. bony fistula in the posterior canal was covered with thin gold leaf and the cavity filled with paraffin. Ten months after the operation tone 512 showed a gain of 15 decibels,

15. Nasiell, W.: Modern Treatment of Otosclerosis, Svenska läk.-tidning. **37:** 137-145 (Jan.) 1940.

16. Holmgren, G.: Four Cases of Operated Otosclerosis, Nord. med. (Hygiea) **4**:3463 (Nov. 25) 1939; Presentation of Four Cases Operated upon for Otosclerosis, Acta oto-laryng. **28**:443, 1940.

1024 a gain of 5 decibels and 2048 no change, an average gain for these speech frequencies of 7 decibels. In case 4 a conservative radical operation was followed three months later by the making of a fistula 3 mm. long in the posterior vertical canal. Thirteen months after operation there was a gain of 15 decibels for 512, of 25 decibels for 1024 and of 20 decibels for 2048, an average gain of 20 decibels for these speech frequencies.

[Comment: These results are the best so far reported by Holmgren and are impressive. The important question of what proportion of patients operated on by his technic show favorable results four to thirteen months after operation is not answered by these articles.]

Canfield [17] reviews the indications for fistulization of the labyrinth in chronic deafness and presents 3 cases, 1 that of a patient whom he considered suitable for operation and 2 those of patients whom he considered unsuitable. One of the latter had long-standing headaches of undetermined origin, which the author now regards as a contraindication; the other had a healed cavity from radical mastoidectomy for chronic suppurative otitis media. These two unsuitable patients showed no improvement in hearing. The suitable patient showed an average gain of 15 decibels for the three conversational frequencies one month after operation.

Fowler Jr.[18] reviews the surgical measures proposed for the improvement of hearing in chronic deafness, these being Hughson's round window operation, Dixon's operation making a permanent postaural fistula into the antrum and fenestration of the labyrinth. The author compliments Hughson on the careful detailed report of his cases, while expressing doubt as to the experimental basis of the operation and the significance of the hearing gains reported. Dixon's operation is criticized for being contrary to the known physiology of the skin in that the placing of a long tube of epithelium, often containing hair, in the mastoid can be expected to cause trouble sooner or later. The same mechanical result could be obtained by the ordinary modified radical mastoidectomy with the usual meatal flap. The author concludes that one should certainly have multiple accurate anteoperative and postoperative audiometric records before accepting any procedure which seems theoretically so unsound.

In the author's opinion fenestration of the labyrinth so far is the one surgical technic which appears promising for the alleviation of conduction deafness. While in France in 1937 and 1938 he saw 39 of

17. Canfield, N.: The Type of Deafness Which Responds to Fistulization of the Otic Capsule, Ann. Otol., Rhin. & Laryng. **49**:248 (March) 1940.

18. Fowler, E. P., Jr.: Critique of Surgical Treatment for Deafness, Canad. M. A. J. **43**:546 (Dec.) 1940.

Sourdille's patients on one occasion and 14 on a second visit. In all but 7 of these 53 patients the fistula test was positive. There seemed no question that there were many cases in which hearing improvement appeared to be excellent, as judged by the whispered voice, but unfortunately, without an audiometer the testing methods used by Sourdille were very crude. The Lempert technic is a modification of Sourdille's, but the author questions the advantage of the endaural over the postaural approach.

On the basis of experimental work by himself and others the author discusses the causes of closure of labyrinthine fistulas. The first factor is bone chips, which act as foci from which new bone growth takes place. Shambaugh Jr. is cited as using a continuous flow of saline solution to remove bone dust and keep it out of the fistula, and Holmgren, as using microsuction. In addition, the author states the belief that higher magnification than that secured by a Zeiss loupe is desirable.

Second, it has been found that the enchondral bone of the labyrinthine capsule does not participate in the bony healing of fractures. Therefore, the fistula should be made in the solid angle, where the enchondral capsule is relatively thick and the periosteal capsule is relatively thin. Since the endolymphatic system is attached to the outer periphery of the canal, it will be less likely to be injured by the fistula in the solid angle.

Third, a larger fistula is apparently less likely to close.

Fourth, infection undoubtedly tends to increase bone regeneration.

Fifth, the author believes that the source of the skin used to cover the fistula is less important than that the skin be thin.

Finally, he expresses the belief that fistulization is as dangerous as any major surgical procedure on the mastoid, perhaps more so, and by comparing the available data of Lempert, Shambaugh Jr., Campbell and Greenfield, he deduces that the chances for a significant hearing improvement lasting six months or more are roughly less than one in three.

[Comment: The author's enumeration of the causes of bony closure is interesting and instructive. His evaluation of the various surgical technics is conservative and not overenthusiastic; at the same time an open mind is maintained toward the future possibilities of the fenestration operation.]

Guggenheim and Guggenheim[19] criticize the fenestration operation on three counts. First, they state that otosclerosis is not only an osseous dystrophy producing an obstruction to sound conduction but that it is

19. Guggenheim, L. K., and Guggenheim, P.: Artificial Fistula in Cases of Otosclerosis, Arch. Otolaryng. **32**:1 (July) 1940.

also a disease of the eighth nerve producing perception deafness, so that even though a fistula operation should prove successful, it could in no way inhibit further deterioration of the neural mechanism. Second, they state that medical treatment of otosclerosis through dietary regulation and the administration of calcium, phosphorus and vitamins has produced improvement in hearing far greater than has any fistula operation thus far reported. Third, they state that "the ingenious difficult and time-consuming technic has nothing whatever to do with the success or failure of the fistula operation" and that the old and simple postaural approach and covering of the fistula with any membrane at hand will work as well and take one sixth the time.

To determine whether or not the burnishing burr inhibits reformation of bone as compared with a sharp instrument, the authors made holes in the skulls of rabbits, using respectively a sharp chisel, a burnishing burr and a burnishing burr plus electrocoagulation to the margins. Eighteen days later, lively and widespread new bone formation was seen where the fistula had been made with the sharp instrument or with a burnishing burr, but no evidence of osteogenesis was seen following electrocoagulation. Two months and two days after operation the most patent fistula was that made with the sharp instrument. Next came the fistula made with the burnishing burr and completed by electrocoagulation, while that made with the burnishing burr alone showed the largest amount of new bone formation. In the opinion of the authors the fact that these operations were performed on a type of bone different from that of the labyrinthine capsule in no way affects the experiment, since all new bone formation is by the membrane-bone process.

Shambaugh [20] takes issue with certain statements made by Guggenheim and Guggenheim relative to the fenestration operation for otosclerosis—namely, that greater improvements have been secured by dietary management and that the intricate and difficult technic has nothing to do with the end results of the operation. Pointing out that no treatment for deafness can be regarded as of proved value until audiometrically substantiated and sustained hearing improvements of more than 10 decibels for the conversational frequencies are reported and that such improvements have never been reported following any medical or surgical treatment except the fenestration operation, the author then presents his own results in 31 cases of otosclerosis in which he operated by Lempert's fenestration technic over a period of slightly more than two years. Of the 31 patients, 6 of whom had had revisions, 24, or 77 per cent, showed an audiometric improvement of more than

20. Shambaugh, G. E., Jr.: Operative Treatment of Otosclerosis: Reply to a Recent Criticism, Arch. Otolaryng. **32**:927 (Nov.) 1940.

10 decibels for the conversational frequencies, three weeks to fourteen months after operation, as follows:

Case	Comment	Time After Operation	Average Change for Speech (512, 1024, 2048) in Decibels
1	Both ears operated on; both fistulas closed; revision of first (left) ear	8 mo.	+37 (gain)
2	Membranous labyrinth injured	.....	No change
3	Serous labyrinthitis	.....	— 5 (loss)
4	Fistula closed	.....	No change
5	Fistula partially closed; revision	1 mo.	+ 8 (gain)
6	Fistula closed	.....	No change
7	Both ears operated on; both fistulas partially closed; revision of second (right) ear	5 mo.	+30 (gain)
8	Right ear	14 mo.	+14 (gain)
	Left ear	2 mo.	+29 (gain)
9	Fistula partially closed; revision	2 mo.	+14 (gain)
10	Fistula partially closed	.....	No change
11	.....	10 mo.	+13 (gain)
12	.....	8 mo.	+14 (gain)
13	Fistula partially closed; revision	4 mo.	+21 (gain)
14	.....	7 mo.	+24 (gain)
15	.....	7 mo.	+11 (gain)
16	Fistula partially closed; revision	2 mo.	+28 (gain)
17	.....	6 mo.	+34 (gain)
18	.....	5 mo.	+11 (gain)
19	.....	4 mo.	+25 (gain)
20	.....	3 mo.	+27 (gain)
21	.....	2 mo.	+14 (gain)
22	.....	2 mo.	+21 (gain)
23	.....	2 mo.	+36 (gain)
24	.....	2 mo.	+21 (gain)
25	.....	2 mo.	+19 (gain)
26	.....	6 weeks	+27 (gain)
27	.....	1 mo.	+ 8 (gain)
28	.....	1 mo.	+32 (gain)
29	.....	1 mo.	+28 (gain)
30	.....	2 weeks	+24 (gain)
31	.....	2 weeks	+25 (gain)

Campbell [21] discusses the selection of patients for the fistulization operation and states that if the hearing is to be brought to a practical level the preoperative loss should not exceed 60 or 65 per cent of conversation, while patients with a loss of less than 35 or 40 per cent probably should not be operated on, because they still have some serviceable hearing, which might be made worse if the operation is unsuccessful. On the other hand, the operation may be justified in lesser degrees of hearing loss when tinnitus is severe or the progressive loss is rapid, since the operation aims to lessen tinnitus and to prevent further loss as well as to improve hearing. Good bone conduction is of extreme importance; there should probably be normal vestibular function, and the skin of the external auditory canal should be healthy. While a normal drum membrane is ideal for operation, it is likely that good results may be obtained with a thickened or scarred drum membrane.

The author emphasizes the importance of adhering closely to the exacting operative technic of Lempert, which requires anatomic knowledge of the region, skill and care in handling of the most delicate

21. Campbell, E. H.: Results in the Labyrinth Fistulization Operation for Chronic Deafness, Ann. Otol., Rhin. & Laryng. **49**:447 (June) 1940.

structures, patience and physical endurance. Proper creation of the fistula determines the success of the operation more than any other stage of the technic.

The author presents a follow-up report of 8 cases previously reported. The average gain for the three speech frequencies (512, 1024 and 2048) was as follows:

Case	Comment	Time After Operation	Change in Hearing: Ear Operated On	Change in Hearing: Ear Not Operated On
1	.....	14 mo.	+27 db.	Slight further loss
2	Membranous labyrinth perforated.....	.....	Considerable reduction	
3	Fistula closed and hearing gain lost...	3 weeks	Final hearing 16.5% below preoperative level	
4	Fistula closed; revised.....	8 mo. (after revision)	+5%	
5	Fistula made in cavity from previous radical mastoidectomy	.....	.....	Further loss of 9%
6	.....	9 mo.	+25 db.	Not stated
7	.....	1 yr.	Further loss of 20%	
8	.....	6 mo.	+21.9%	

Lempert,[22] in a complete and detailed account, reviews his observations and results in 120 cases in which operation was done by his one stage technic. The essentials of this technic are:

1. Creation of a new window in the horizontal semicircular canal to replace the occluded oval window.

2. Reconstruction of the tympanic cavity to include within it this new window by:

(*a*) Creation of an intact continuous membrane of the skin of the external canal, Shrapnell's membrane and the drum membrane.

(*b*) Amputation of the head and neck of the malleus, to permit Shrapnell's membrane to be pulled back to cover the new window.

(*c*) Preservation of the incus in its normal position.

(*d*) Removal of the pyramidal eminence, to widen the tympanic cavity.

(*e*) Closing the tympanic cavity from the outer ear with the tympanomeatal membrane, so as to include the new window in the tympanic cavity.

The degree and permanence of the hearing improvement are directly proportional to the exactitude and finesse with which each step in the

22. Lempert, J.: Endaural Fenestration of the External Semicircular Canal for Restoration of Hearing in Cases of Otosclerosis: Summary Report of One Hundred and Twenty Cases, Arch. Otolaryng. **31**:711 (May) 1940.

technic is accomplished. The largest possible window is made in the horizontal canal, injury to the delicate membranous canal being avoided.

The operation is indicated in the following conditions: 1. Bilateral progressive loss of hearing. 2. Fixation of the stapes with a normal condition of the round window. 3. Loss of hearing by air conduction sufficient to make practical hearing for conversation impossible, while the bone conduction for the conversational frequencies remains normal or shows a loss not greater than 30 decibels. 4. Normal state of the tympanic membrane. 5. Complete absence of middle ear infection. 6. Patency of the eustachian tubes. 7. Normal state of health.

The following data are recorded as to surgical accidents in 120 cases: 1. In 1 case the tympanomeatal membrane was accidentally torn. The meatal skin was used to cover the fistula. 2. In 1 case an accidental perforation of the drum membrane healed, with a good hearing result. 3. In 1 case fracture of the facial canal resulted in a temporary facial weakness. 4. In 3 cases dislocation of the incus necessitated its removal. 5. After accidental puncture of the sigmoid sinus, the operation was completed with a good result.

The following postoperative observations are recorded: 1. In no case did the postoperative temperature exceed 1 degree above normal. 2. In all cases vertigo and nausea lasted one to three days. 3. In 2 cases postoperative serous labyrinthitis resulted two to three days after operation in complete loss of cochlear and vestibular function. 4. In 1 case necrosis of a small area of the mastoid cortex delayed healing. 5. One patient died of coronary thrombosis while dressing to go home from the hospital. 6. In no case was infection of the outer, middle or inner ear encountered. 7. No subjective symptoms from the open fistula were noted after the third postoperative week.

The hearing results in the 120 cases were: permanent restoration of physiologic hearing, 69; marked improvement but not enough to serve as practical hearing, 10; no improvement, 27; further loss, 14.

Tinnitus was completely relieved in all 69 of the patients who experienced good results and was diminished in the 10 who experienced fair results. Tinnitus was unchanged when the hearing was unchanged and was worse when the hearing was worse.

In 100 of the 120 cases the new window remained open. In 31 cases revisions were done in 1 case four times and in 4 cases twice.

In 19 cases the positive character of the fistula test disappeared and the hearing improvement receded to the preoperative level. In 14 of these the fistula was completely closed by new bone formation; in 3 cases the fistula was largely but not entirely closed by new bone, while in 2 cases the fistula was filled with dense fibrous tissue, adherent to the membranous labyrinth.

In 4 cases the fistula test remained positive but the hearing improvement disappeared. Revision showed a fistula still open but much decreased in size because of new bone formation.

In 2 cases revision was done because the original fistula was small and no improvement in hearing resulted. No new bone formation was found, and so the fistula was enlarged.

In 5 cases the hearing had receded to the preoperative level and the fistula test showed a very sluggish response. In 4 of these the membranous labyrinth was found to be inflamed, thickened and adherent to the endosteum, and in 1 a spicule of bone was found on the membranous labyrinth.

In 1 case the tympanomeatal membrane sloughed, the hearing rapidly receded and the fistula test became negative. Revision showed fibrous tissue growing into and filling the perilymph space from the mastoid cavity.

The best results in revisions occurred when a thin Thiersch skin graft was used to line the inner surface of the tympanomeatal membrane, so that skin rather than periosteum was placed over the fistula. The results in the 39 revisions in 31 cases were: permanent improvement in hearing, 11; hearing temporarily improved but lost again, 12; hearing unimproved or less than a practical level, 5; postoperative serous labyrinthitis with further loss of hearing, 10.

[Comment: Lempert is criticized, partly by those who have not taken the trouble to investigate and observe his work and who have perhaps seen one or two poor results, since patients with poor results are more prone to seek help from other physicians than are those with good results. Others criticize Lempert because of his enthusiasm for his operation. For example, he uses the term "permanent improvement" when one cannot yet be sure of the ultimate fate of the hearing in the cases in which his technic has been used, since insufficient time has elapsed. Exception can also be taken to certain statements; i. e., that Shrapnell's membrane reaches and covers the fistula and that infection of the outer middle or inner ear was never encountered. I believe that a slight low grade infection of the postoperative cavity must be a common occurrence and that Shrapnell's membrane does not cover the fistula. My experience with tinnitus has been less favorable. In some patients with excellent and sustained hearing improvement the tinnitus has recurred, and in others it has not been relieved. In other respects my experiences with the fenestration operation are similar to Lempert's. Recently, I have had the opportunity of reviewing the complete audiometric records from all of his cases, good and bad, and I can verify the authenticity and reliability of his published results. There can be no question that Lempert has made a monumental contribution to otology in continuing the pioneer work of Passow, Bárány, Jenkins, Holmgren

and Sourdille and in devising a practical one stage operation—the first measure in the history of otology which offers the patient with otosclerosis the reasonable possibility of recovering much of his lost hearing.]

CHRONIC ADHESIVE DEAFNESS

Guild and his associates [23] examined 1,365 Baltimore school children between the ages of 8 and 14 during the school year of 1939-1940. The threshold of hearing for frequencies 32, 64, 128, 256, 512, 1024, 2048, 2896, 4096, 5793, 8192, 10,331, 13,004 and 16,384 was determined in a sound-proof room, and the nasopharynx was inspected with a nasopharyngoscope. Of the total number of children examined, treatment was advised for 239 and begun for 208. At the time of writing 184 had been reexamined one to six times each; each had had one to nine treatments, and the time elapsed since the treatment was begun was two to six months. It was therefore too early to draw final conclusions. Already, however, the adenoids of 95 of the 184 children were definitely smaller, as the result of radium treatment in the nasopharynx, and in many the shrinkage of lymphoid tissue was truly remarkable. Eustachian orifices that were completely overgrown with lymphoid tissue at the first examination were then free from any obstruction.

The control group consisted of 467 children who had no treatment but were reexamined three to four months after their first examination. Most of these had large adenoids but only slight or no impairment of hearing. At the second examination the adenoids were smaller than at the first examination in only 19.1 per cent, compared with 51.6 per cent for the children who were treated.

Of the entire 1,365 children examined, only 58.8 per cent showed normal hearing in both ears. This figure differs greatly from those recorded in previous surveys of the hearing in children because no previous study included testing with so many of the high frequencies.

Crowe [24] stated that adenoids recur in more than 75 per cent of children whose tonsils and adenoids have been removed before the age of puberty and that this recurrence often interferes with the ventilating function of the eustachian tubes and leads to an insidious painless type of progressive deafness. The changes in the middle ear are, first, hyperemia, then a secretory stage, gradually becoming myxomatous, and finally the formation of fibrous tissue, which interferes more and more with the mobility of the ossicles. These changes are apt to occur in the attic, visible in Shrapnell's membrane. Impairment of hearing in

23. Guild, S. R.; Polvogt, L. M.; Sandstead, H. R.; Loch, W. E.; Langer, E.; Robbins, M., and Parr, A.: Impaired Hearing in School Children, Laryngoscope **50**:731 (Aug.) 1940.

24. Crowe, S. J.: The Recognition, Treatment and Prevention of Hearing Impairment in Children, Laryngoscope **50**:658 (July) 1940.

children, whatever the primary cause, is almost invariably complicated to a greater or less degree by partial obstruction of the eustachian tubes secondary to hypertrophy of the lymphoid tissue in the nasopharynx.

The patency of the eustachian tubes in children is not determined by inflation, which is an artificial procedure, but by inspection of the tympanic membranes for retraction (especially Shrapnell's membrane), by examination with the nasopharyngoscope and by voice and audiometric hearing tests. Regardless of the primary cause of the deafness, every child with evidence of obstruction of a eustachian tube should have something done to restore and maintain the patency of the tube.

In conclusion, the author stresses the common occurrence of adenoids in children, the presence of which may lead to chronic progressive deafness and which are so located around the pharyngeal orifices of the eustachian tubes that they cannot be removed surgically. The dosage of radium employed for this lymphoid tissue is far below the amount that could cause any irritation or injury to the mucous membrane or surrounding structures, but radium must be administered carefully and never by a technician. Radium should never be given in the presence of acute nasopharyngitis. It is of no value in the treatment of chronic suppurative otitis media, otosclerosis or nerve deafness but is to be used for the sole purpose of restoring the normal ventilating function of the eustachian tubes.

Burnham [25] describes the technic employed by Crowe and his associates for irradiation of lymphoid tissue in the nasopharynx. The nasal applicator is 15 cm. long. On the tip there is a brass tube, into which may be placed radon or radium. The average dosage is 2 gram minutes. With this dosage, at least six weeks should intervene between applications. There is great variation in the vulnerability of normal hyperplastic lymphoid tissue to radiation. The repetition of the treatment should depend on the hearing and on the appearance of the nasopharynx.

[Comment: That the type of conduction deafness due to enlarged adenoids, namely, secretory otitis, may be associated with as much or more of a loss for high tones than for low tones was first pointed out by Shambaugh Jr. at the meeting of the American Otological Society in 1936 and has since been confirmed by Crowe, Guild and their associates. It has long been known that the most frequent cause for impairment of hearing in children is secretory otitis, the result of tubal occlusion from enlarged adenoids. It is a common observation that recurrence of adenoid tissue is frequent in children. Members of the Baltimore group have reemphasized these facts. They have offered a new method for reducing this lymphoid tissue—namely, the application of

25. Burnham, C. F.: Irradiation Treatment of Hyperplastic Lymphoid Tissue, Laryngoscope **50**:663 (July) 1940.

radium to this tissue in the nasopharynx—and they have discovered a surprising incidence of high tone loss in children. How large a proportion of this high tone loss is attributable to tubal occlusion and will be recovered after irradiation of the nasopharynx has not yet been determined.]

Holmgren [26] ligated the eustachian tube in dogs and was able to demonstrate negative pressure in the middle ear and typical noninflammatory transudation into the middle ear.

[Comment: To help settle the matter of whether or not prolonged tubal occlusion can lead to permanent fibrotic changes in the middle ear with chronic adhesive deafness, the author should repeat his experiments, with histologic study of the middle ear at longer periods after the occlusion.]

Maxwell and Richter [27] examined 50 patients who had undergone radical mastoidectomy, to determine the effect on the hearing. Of patients showing a preoperative loss of 18 to 30 decibels, a further loss followed operation in 72.7 per cent, while of those showing a preoperative loss of 60 to 70 decibels there was an improvement after operation in 60 per cent. In the entire series the hearing was made worse as often as it was improved. A dry healed cavity was present in 84 per cent, while 16 per cent showed some mucoid discharge from the region of the tube.

Ruskin [28] advises the use of the gold-cut dental cylinder used for small dental fillings, to close perforations of the drum membrane. The cylinder is held with a bayonet forceps, is gently flattened with a second pair of forceps, quickly flamed for sterilization and placed over the defect, being forced gently into the perforation so that the edges grip the rim of the perforation. Contact between the gold and the promontory is advantageous. The advantages of the spongy gold cylinder are that it is flexible, soft, easily molded, yet light and permanent in form, sterilizable and nonirritating to the tissues. It tends to stimulate epithelization and closure of large perforations. Of 18 cases the hearing was markedly improved in 56 per cent, moderately improved in 38 per cent and unimproved in 6 per cent, and in 2 cases the perforation was closed or closing. Prior to application of the prosthesis, all suppuration must have ceased.

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26. Holmgren, L.: Experimental Tubar Occlusion, Acta oto-laryng. **28**:587, 1940.

27. Maxwell, J. H., and Richter, H. J.: Radical Mastoidectomy: Its Effect on Hearing, Arch. Otolaryng. **31**:426 (March.) 1940.

28. Ruskin, S. L.: Gold Prosthesis in Drum Defects, Laryngoscope **50**:573 (June) 1940.

LABYRINTHINE DEAFNESS

Traumatic Nerve Deafness.—Campbell and Hargreaves [29] classify aviation deafness as: 1. Acute fatigue of the end organ and related structures. 2. Chronic accumulative fatigue of the end organ and related structures. 3. Conduction deafness due to changes in pressure in the middle ear. 4. Chronic conduction deafness due to alteration in tissue resulting from faulty ventilation of the middle ear.

To note the effect of acute fatigue, one of the authors flew for six one hour periods on different days and found each time a 10 decibel loss confined to the 4096 tone. An average of one hour and thirty-four minutes was required for complete recovery. After two hour periods of flight on six successive days the dip involved also the 2896 tone and was greater for 4096, averaging 18 decibels' loss. The time required for complete recovery averaged four hours and fifteen minutes. In the third experiment the hearing was tested after four periods of four hours' flight each, and the dip broadened to include 2048, 2896, 4096 and 5792, with an average loss for 4096 of 22 decibels. Complete recovery required an average of six hours.

It was observed that the depth of the 4096 dip varied with the type of plane and the noise of the engines. The dip at 4096, when most of the noise lies below 1,000 vibrations, is perhaps due to anatomic considerations, the region in the cochlea that responds to 4096 being closest to the oval and round windows and thus more directly exposed to the vibratory energy.

Chronic fatigue was evidenced by a permanent notch or dip in the 4096 area and first appeared after about two hundred and fifty hours of flying in an open cockpit. Much greater losses occur, however, after only a few hundred hours of flight as a result of excessive noise or with a more susceptible person.

Acute deafness due to failure to aerate the middle ear during rapid descent shows a loss for low tones only and is of the pure conduction deafness type.

Chronic conduction deafness due to inadequate aeration of the middle ear shows the usual picture.

The authors believe that losses of hearing in those who fly should decrease in the future with improvements in aircraft design and with the employment of constant pressure cabins and soundproofing.

[Comment: The conception of acute and chronic auditory fatigue with the characteristic 4096 dip is one of the new additions to otologic knowledge and is attributable to the audiometer with its accurate testing for higher tones.]

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29. Campbell, P. A., and Hargreaves, J.: Aviation Deafness—Acute and Chronic, Arch. Otolaryng. **32**:417 (Sept.) 1940.

Hurst [30] believes, as a result of extensive experience with functional deafness in soldiers during the last war, that in many cases of deafness among civilians there is a functional element, and also perhaps in a small proportion of cases of deaf-mutism among children.

The usual history of functional deafness in soldiers is that of initial organic deafness resulting from the noise and concussion produced by the nearby explosion of a shell of high power. This initial deafness may be due to a concussion of the inner ear, a rupture of the drum membranes or perhaps a dislocation of the ossicles. Normally this initial deafness passes off in a few hours or in a few days. If it persists longer, it is usually hysterical, at least in part. Probably the initial concussion deafness makes such an impression on the soldier that he convinces himself by autosuggestion that he is permanently deafened. This is more likely to occur in those who have had previous aural disease and are "hearing conscious." In hysterical deafness the patient apparently ceases to listen and as a result of inattention fails to hear, exactly as one may fail to hear even the sound of the preacher's voice during a dull sermon.

The diagnosis of hysterical deafness is made on the following points:

1. History. Complete bilateral deafness following the explosion of a powerful shell in the previous war was generally hysterical.
2. Other evidences of hysteria may be present but have often been absent.
3. Hysterical anesthesia of the external ear, supposed to be found in hysterical deafness, is not a reliable sign.
4. The auditory motor reflex is present in malingerers but is absent in persons with total organic or total hysterical deafness.
5. Hysterical deafness persists during sleep.
6. Hearing does not return during hypnosis.
7. The voice in total hysterical deafness remains unaltered, whereas in total organic deafness it changes in character.
8. Lip reading is learned with remarkable rapidity in hysterical deafness. A malingerer is unlikely to learn lip reading.
9. Normal vestibular responses with total bilateral deafness nearly always mean hysterical deafness, since total bilateral organic deafness would nearly always be associated with deficiency or loss of the vestibular responses.

The treatment of hysterical deafness was formerly chiefly by various forms of suggestion, but it was found that a more rational form of psychotherapy—namely, explanation, persuasion and reeducation—gave

30. Hurst, A. F., and Miller, E.: Discussion on Functional Deafness, J. Laryng. & Otol. **55**:393 (Aug.) 1940.

better and more lasting results. The patient is made to understand by written explanations how he became deaf and how the original cause of his deafness has now disappeared; as the deafness was at first organic, he could not hear, however much he listened, and, consequently, after a time he ceased to listen. He is then persuaded to listen intently and is taught that listening is just as active a process as moving and requires a conscious effort on his part until it becomes automatic once more.

[Comment: My one experience with hysterical deafness concerned a student nurse who apparently was unsuited for her new profession but was unwilling to admit it to herself or her family. The usual tests for malingering were negative; the auditory motor reflex was absent (she did not blink the eyes with a sudden loud noise close to the ears); the audiograms showed only a slight remnant of hearing, and the same audiogram was obtained on successive tests. The diagnosis of hysterical deafness was made on the basis of the facts that she had become a proficient lip reader within a few months and that her voice had not changed. Autosuggestion produced by having the patient inhale carbon dioxide until a slight convulsion occurred resulted in an immediate and dramatic restoration of normal hearing. The hysterical deafness recurred, however, and a permanent cure did not result until she gave up her nursing school course. In my experience hysterical deafness is rare in civilian life.]

Nerve Deafness from Infection.—Rodger [31] summarizes his observations in 500 cases of syphilis of the ear, nose and throat encountered over a ten year period, in which the diagnosis was confirmed by a positive Wassermann test or unquestioned signs of congenital syphilis (such as interstitial keratitis and Hutchinson teeth).

Hereditary syphilis of the ear occurs in two forms, the early and the late. The early form belongs to the secondary stage of syphilis and results from meningoneuritis affecting the eighth nerve as part of syphilitic basal meningitis or from otolabyrinthitis affecting the periosteum of the middle and inner ear, with or without suppurative otitis media. In both forms the diagnosis is usually made in retrospect. A child is brought for examination because he is not learning to speak. There is a history suggestive of mild meningitis or otorrhea, the hearing is gone and the Wassermann test of the blood is negative.

Deafness occurring later in the course of congenital syphilis belongs to the tertiary stage of syphilis. The deafness is usually symmetric, it comes on as a rule two or three years after the interstitial keratitis,

31. Rodger, T. R.: Syphilis as Seen by the Aural Surgeon, J. Laryng. & Otol. **55**:168 (March) 1940.

there is no associated otitis and the progressive loss of hearing to profound deafness is rapid—often occurring in less than a year after the onset. The involvement is primarily in the labyrinth, with simultaneous involvement of cochlear and vestibular function.

In acquired syphilis there may be otolabyrinthitis or meningoneurolabyrinthitis in the secondary stage, similar to the lesions in the early hereditary form. Since this nerve deafness frequently coincides with the period of active treatment, it has sometimes been attributed to arsenic poisoning. Ramadier believes, however, that the deafness is rather a coincidence and that the danger lies in inadequate dosage and treatment rather than in overdosage.

The most common form of deafness in acquired syphilis is that due to neurolabyrinthitis without otitis media and analogous to that observed in syphilis of the late congenital type. While usually bilateral, in a fourth of the cases studied this form of deafness remained unilateral. Tabes and paresis may be present with the neurolabyrinthitis.

The prognosis as far as hearing is concerned is the poorest possible in syphilitic deafness, because permanent damage has already occurred before treatment is begun, and there seems to be no hope of recovery of function. The author has seldom seen any improvement in the hearing even after prolonged medication, except when there was coincidental catarrhal or suppurative deafness.

[Comment: This is a valuable contribution, based on an unusually large series of cases. Every patient with inner ear deafness should have a serologic examination. Only in this way will syphilitic nerve deafness be detected early enough to arrest the progressive loss of hearing by intensive and adequate treatment. My small experience with the treatment of syphilitic nerve deafness has been more favorable than the author's. In a case of nerve deafness with secondary syphilis the hearing became normal and remained so after treatment. In 2 cases of congenital syphilis the rapidly progressive loss of hearing was arrested by intensive treatment.]

Hagens [32] reports the histologic examination of the temporal bones of a child who died fifty-three days after the onset of epidemic cerebrospinal meningitis and forty-six days after deafness had occurred. Practically all elements of the inner ear had been destroyed and replaced by granulation tissue. The avenues of infection were along the auditory nerve and probably also the cochlear aqueduct. These observations agree with those made in previous histologic studies of similar cases as reported in the European literature.

32. Hagens, E. W.: Pathology of the Inner Ear in a Case of Deafness from Epidemic Cerebrospinal Meningitis, Ann. Otol., Rhin. & Laryng. **49**:168 (March) 1940.

Kelly [33] reports complete loss of cochlear and vestibular function on the left side seven months after an attack of herpes confined to the scalp on the same side. The patient became very dizzy and noticed deafness in the left ear soon after the onset of the herpes. The author concludes that the herpes had affected the left fifth nerve, the spiral ganglion of the auditory nerve, Scarpa's ganglion of the vestibular nerve and the posterior root ganglion of the second and third cervical nerves.

Nerve Deafness from Endocrine Disturbances.—Prietzel [34] states that in presbycusis there are changes in the basilar membrane due to deposition of a connective tissue substance. Using testosterone propionate in 14 cases of presbycusis and tinnitus (intramuscular injection of 25 mg. twice a week), the author achieved "a great improvement of the tinnitus and hearing."

[Comment: As has been repeatedly pointed out in this review, because of the psychologic elements in hearing no report of hearing improvement from therapy can be regarded as proving the value of therapy unless there is an audiometrically recorded improvement of more than 10 decibels. There is no evidence that presbycusis is related to the endocrine glands, and there is no acceptable evidence that the administration of an androgen is of any benefit in presbycusis.]

Selfridge [35] presents the cases of 6 patients with chronic progressive deafness who were treated with pituitary, thyroid and estrogenic substances, some with and others without vitamins. In each case audiometric improvement occurred, amounting to a gain of 20 decibels for the conversational tones in 2 cases, with lesser gains in the others. In each patient there was evidence of glandular dysfunction, usually in the form of abnormal menses. The author concludes that estrogenic substances probably play an important role in conduction deafness, while there is evidence that thyroid, pituitary and adrenal cortex extract are also of importance in treatment.

[Comment: As was pointed out in this review in a previous year, the possibility that deafness results from subclinical avitaminosis would be more acceptable if deafness were known to be a symptom of xerophthalmia, beriberi, pellagra, scurvy, rickets or other vitamin-deficient states. Similarly, the possibility that deafness is due to mild endocrine imbalance would seem more logical if marked endocrine deficiencies were known to result in deafness. As far as I know,

33. Kelly, D. B.: Deafness and Loss of Vestibular Function Following Herpes of the Scalp, J. Laryng. & Otol. **55**:286 (June) 1940.

34. Prietzel, F.: Zur Keimdrüsenbehandlung bei Alterschwerkörigheit, Monatschr. f. Ohrenh. **74**:18 (Jan.) 1940.

35. Selfridge, G.: Chronic Progressive Deafness with Special Reference to Estrogenic Substances: A Further Contribution, Ann. Otol., Rhin. & Laryng. **49**:52 (March) 1940.

myxedema is the only disease due to endocrine deficiency which is frequently associated with impairment of hearing, and the impairment may be due partly to the mental sluggishness in this condition as well as to pathologic changes in the peripheral auditory apparatus.]

Nerve Deafness from Avitaminosis.—Covell [36] tabulates the histologic changes in the ears of animals kept on vitamin-deficient diets.

Vitamin A deficiency in rats was associated with middle ear infections, increase in the periosteal layer of the otic capsule and occasionally exostoses in the internal auditory canal, degeneration of the cells of the stria vascularis, external sulcus cells and parts of the organ of Corti, and slight demyelination of the cochlear nerve.

The absence from the diet of vitamin B_1 and various factors of the B_2 complex results in a higher incidence of otitis media. Degenerative changes are found in the external hair cells of animals on a riboflavin-deficient diet. In all avitaminoses of the B group the cells of the spiral ganglion are altered in appearance, though the alteration is less in avitaminosis associated with vitamin B_6. The three most important vitamins for the maintenance of the myelin sheath of the nerve are, in order, vitamin B_6, thiamine and riboflavin. The cochlear nerve is more susceptible to alterations than any other cranial nerve, with the possible exception of the optic.

Vitamin C–deficient guinea pigs reveal hemorrhage into the lining of the middle ear cavity, with an increase in thickness of the submucosa. There is a tendency for cellular marrow to be replaced by fibrous marrow. The cells of the organ of Corti, including the hair cells, are swollen and degenerated, as are also some of the cells of the stria vascularis.

Vitamin D–deficient rats frequently have otitis media. Osteoid is laid down in the bone of the ossicular chain and otic capsule. Rats low in vitamin E show otitis media, fibrous tissue replacement in the muscles of the middle ear, small exostoses and irregularities of the periosteal bone and hemorrhages about the nerve in the modiolus, under the dura and the submucosa of the middle ear.

In discussing these findings the author points out that the field of dietary diseases of the nose and throat remains uninvestigated up to the present time and is perhaps a more promising field for study than the problem of deafness from avitaminoses.

[Comment: Covell's studies are of fundamental importance in the investigation of the relation of vitamins to deafness. It is noteworthy that in every deficiency, changes in the ear were observed, so that not one but all the vitamins should be given in attempts to improve hearing.

36. Covell, W. P.: Pathologic Changes in the Peripheral Auditory Mechanism Due to Avitaminoses, Laryngoscope **50**:632 (July) 1940.

However, workers must be cautious in applying the results of these animal experiments, in which the animals are reduced to a condition often close to death by complete vitamin deficiency, to human beings in the United States, who may suffer from partial deficiencies in one or more vitamins, owing to poor diet or deficient utilization, but who are in comparatively good health and are therefore not comparable to animals on a totally deficient diet. Though my own experience with vitamins in the treatment of deafness has been completely negative to date, I optimistically continue to prescribe large doses of all the vitamins to patients with progressive nerve deafness of unknown etiology.]

Childrey [37] points out that if nerve deafness can be largely explained by a deficiency of vitamin B, it seems that there should be other evidence of such avitaminosis. Nevertheless, because vitamin B seems harmless and for lack of something better, the author used it for over a year in the treatment of many patients with nerve deafness and with combined deafness with a large neural element, when no other cause for the hearing impairment could be demonstrated. Thiamine hydrochloride, as well as the entire vitamin B complex, was given orally in doses varying from 300 to 1,200 units daily, and thiamine hydrochloride was given subcutaneously in 3,000 to 5,000 unit doses. Nearly every patient was given a list of foods rich in vitamin B and advised to use these freely. Audiograms made after treatment in 26 cases showed no hearing gain in any case. Thiamine hydrochloride was also used in many cases of tinnitus, and in only 1 instance did it appear to give relief. In view of his negative results the author expresses the hope that both the dosage and the method of administration of the vitamin were inadequate.

Sato [38] observed that in rats fed a diet poor in vitamin D rickets of the labyrinth bone developed in addition to rickets of the long bones. At the same time, degeneration of the spiral ganglion cells of the acoustic nerve occurred, as is seen in clinically advanced cases of rickets.

[Comment: The author's statement that advanced rickets is associated with nerve deafness is interesting, if true. It is new to me.]

Kobumura [39] gave large doses of vitamin B_1 by injection to a 22 year old patient with labyrinthogenous hardness of hearing and tinnitus, without beriberi, and reports a successful result. In the discussion which followed this report, Chida reported a similar case.

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37. Childrey, J. H.: Vitamin B and Other Measures in the Treatment of Deafness, Laryngoscope **50**:648 (July) 1940.

38. Sato, M.: Ueber Veränderungen des Gehörnervs durch den Mangel an Vitamin D, Okayma-Igakkai-Zasshi **52**:100 (Jan.) 1940.

39. Kobumura, M.: Ein geheilter Fall von Schwerhörigkeit durch vitamin C, Oto-rhino-laryng. **12**:706, 1939.

Baer [40] was unable to obtain improvement in 15 cases of otosclerosis by treatment with vitamin C and vitamin B_1. Although this treatment was unsuccessful, the author suggests trials with improved and newer preparations.

Guggenheim,[41] discussing the advances made in the treatment of deafness during 1939, states that in Ménière's syndrome vitamin B complex should be given orally and intravenously, to prevent neural degeneration. For nerve deafness not due to bacterial invasion of the labyrinth or to acoustic trauma the author advises the removal of foci of infection and the administration of vitamin B complex in large doses: thiamine hydrochloride, 4,000 to 20,000 units daily; nicotinic acid, 100 to 500 mg. daily, and brewers' yeast, 4 to 8 tablets daily. In addition, a diet fully adequate in vitamins A, C and D is given. Treatment should be persisted in for eighteen to twenty-four months.

The author treats otosclerosis by: (1) analysis and regulation of the diet; (2) administration of approximately six times the normal requirement of calcium and phosphorous, and (3) administration of approximately six times the normal requirement of vitamins A, C, D and B complex.

During three years not 1 patient under this treatment designed to reverse halisteresis and neural degeneration has shown further loss of hearing, and a fair percentage have shown definite improvement both in hearing and in general health. The author expresses the belief that the fistula operation has the following disadvantages: uncertainty that the fistula will remain patent, the possibility that intralabyrinthine adhesions will prevent sound waves from passing through when the fistula test is positive and the fact that the operation utterly ignores the neuropathic aspects of otosclerosis, which can produce a progressive loss of hearing even with a patent fistula, thus rendering the fistula useless. Meanwhile, the patient has lost his chance of help through dietary and supplemental vitamin therapy.

[Comment: Guggenheim's enthusiasm for vitamin therapy in nerve deafness, Ménière's syndrome and otosclerosis has not as yet been supported by acceptable evidence that vitamin deficiency is a factor or that large doses of vitamins are beneficial in these conditions. As Bunch pointed out (see the review for 1939), when a group of patients with otosclerosis are followed, in the majority of cases no demonstrable increase in the hearing defect will be noted during periods as long as six years. Therefore, the fact that Guggenheim's patients are not worse

40. Baer, M.: Zur Vitaminbehandlung der Otosclerose, Monatschr. f. Ohrenh. **74**:113 (March) 1940; L'oto-sclérose est-elle influençable par les vitamines, Rev. de laryng. **61**:33 (Jan.) 1940.

41. Guggenheim, L. K.: Treatment of Deafness, Laryngoscope **50**:773 (Aug.) 1940.

after three years on his dietary management does not prove the therapeutic value of this treatment. If he has observed definite improvement in hearing greater than the normal range of variation, he should present audiometric proof of this. Should the evidence be forthcoming that dietary management of otosclerosis will arrest or prevent the nerve degeneration, as all otologists sincerely hope will be the case, there is nothing to prevent the use of vitamins, calcium and phosphorus after the hearing for air-borne sound has been partially restored by fenestration.]

Selfridge [42] reviews the relation of vitamins to general health and to susceptibility to infections and concludes that an optimum diet containing all the vitamins reduces materially the tendency to catch colds.

Veasey [43] reviews the literature on vitamin B and presents 7 cases of deafness and tinnitus in which treatment with vitamin B complex from rice bran, containing 1,000 international units of B_1 and 200 Sherman-Bourquin units of B_2, was given daily. In 3 cases slight improvement in hearing occurred as measured by the duration of hearing for tuning forks; in 4 cases there was no change. The author believes that these cases suggest that there is a possibility of helping some deaf patients by this means.

[Comment: The improvement noted is so slight as to be within the normal limits of variation.]

Baer [44] notes that a reducing diet is usually low in calories but high in vitamins and that loss of weight usually results in a lowering of blood pressure. Since tinnitus and hypertension are known to be related, the alleviation of tinnitus in patients on a high vitamin diet may be due to lowering of blood pressure as well as to administration of vitamin C. However, the author believes that administration of ascorbic acid often terminates tinnitus, and that when this is combined with the administration of vitamin B_1 the results are still more successful.

Treatment of Nerve Deafness.—Houser, Campbell and Schluederberg [45] treated 56 patients with chronic deafness of one to eighteen years' duration, all but 6 of whom had tinnitus. All the patients were observed and treated over a period of two to three months, 1 cc.

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42. Selfridge, G.: Nutrition and the Ear, Ann. Otol., Rhin. & Laryng. **47**:674 (Sept.) 1940.

43. Veasey, C. A.: Nature of Vitamin B and Its Components, with Special Reference to Nerve Deafness, Arch. Otolaryng. **31**:74 (Jan.) 1940.

44. Baer, M.: Die Möglichkeiten der Vitamin-C-Behandlung bei Innenohrschwerhörigkeit und Ohrensausen, Monatschr. f. Ohrenh. **74**:342 (July) 1940.

45. Houser, K. M.; Campbell, E. H., and Schluederberg, H.: The Treatment of Chronic Deafness and Tinnitus Aurium with Prostigmine, J. A. M. A. **115**:994 (Sept. 21) 1940.

of 1:2,000 solution of prostigmine methylsulfate being given subcutaneously every third day, with 15 mg. of prostigmine bromide by mouth after each meal during the first six weeks of treatment. Each case was studied carefully before treatment was begun, all cases of middle ear or tubal inflammation being excluded. During treatment audiograms were taken every two to three weeks. There were 29 cases of perceptive deafness, 19 of conduction deafness and 9 of mixed deafness. An improvement in hearing of 10 decibels or more occurred in the right ear in 12.5 per cent and in the left ear in 7.1 per cent, but of the 7 patients whose hearing was thus improved only 3 were able to notice the improvement, and 1 of them had syphilitic nerve deafness, which abated only when antisyphilitic treatment was given. Tinnitus was alleviated in 7 patients. In 3 improvement was doubtful and in 6 there was definitely nó improvement. The authors conclude that prostigmine is of little if any value in the treatment of tinnitus and deafness.

[Comment: The authors are to be complimented on the careful manner in which their study was carried out: Cases of deafness due to self-limited conditions (acute tubal and middle ear inflammation) were excluded; the hearing was followed audiometrically; the number of patients treated was large. Their study would have been even more valuable had they followed for the same period an equal number of patients treated with sterile hypodermic injections as controls. A great deal of useless and unnecessary treatment would be avoided if all investigators would properly follow and control their work before announcing a new treatment for deafness.]

Miscellaneous Aspects of Nerve Deafness.—Fowler [46] distinguishes two types of tinnitus, the "vibratory," which is easily masked by pure or mixed tones and is conceived of as due to a lesion of the basilar membrane, and the "nonvibratory," which is masked with difficulty and is conceived of as due to a central neural lesion. The author has observed that when tinnitus accompanies a sharp dip or drop in the audiogram it is usually confined in pitch within the limits of this dip, or drop, and that when it accompanies a flat loss of hearing, it is a mixed noise including all tones. In many cases the loss of hearing seems to be due in part at least to the masking effect of the tinnitus itself—when the tinnitus disappears, the hearing improves or recovers to a normal level. Deafness may occur without head noises, but head noises cannot occur without deafness. Tinnitus is a symptom of aural trouble and a danger signal of trouble ahead. The author suggests that many treatments which appear to improve hearing, such as the fenestra-

46. Fowler, E. P.: Head Noises: Significance, Measurement and Importance in Diagnosis and Treatment, Arch. Otolaryng. **32**:903 (Nov.) 1940.

tion operation, do so because of a coincidental alleviation of the head noises.

[Comment: Considering the frequency of the symptom, practically nothing is known of the cause or the cure of tinnitus except that the condition is often, but not always, present in hearing impairment and that it usually is abated if the hearing can be improved. There is no specific treatment for tinnitus that is of any avail except to remove the cause, if possible. The fenestration operation may relieve tinnitus in cases of otosclerosis, but the hearing improvement cannot always be ascribed to the relief of tinnitus, since the improvement in hearing occurs when there is no tinnitus or when the tinnitus is not improved. The author's contribution represents an interesting and unique scientific analysis of the troublesome symptom of tinnitus.]

Asherson [47] suggests that since nerve deafness frequently is manifested by a loss of hearing for high tones, the low tones remaining normal, the cochlear nerve consists of two trunks, one for high and one for low tones, each with a separate arterial blood supply. The 4096 dip of occupational deafness may be due to spasm and later to occlusion of the end artery which terminates at this point in the cochlea. A case of acute nerve deafness in which first the hearing for high tones, later that for low tones was affected, with recovery of the hearing for high tones first and of that for low tones later, is presented as evidence of this blood supply by two arterial branches to two trunks of the cochlear nerve.

[Comment: The author's hypothesis does not seem consistent with clinical observations or with anatomic studies. His single case report can hardly be regarded as proof of his theory.]

Shambaugh [48] defines diplacusis as the hearing of one sound as two. The three varieties of diplacusis are: echo diplacusis, in which the sound is heard a fraction of a second later in one ear than in the other; monaural dysharmonic diplacusis, in which a single tone is heard in one ear as a double tone of two pitches, and binaural dysharmonic diplacusis, in which a single tone is heard at a different pitch by the two ears. This last variety is by far the most common. It is easily tested for by tuning forks, the test fork being placed before each ear alternately and at such a distance that the sound is heard at the same intensity in both ears. Most patients with diplacusis are not aware of the symptom until they are specifically tested for it.

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47. Asherson, N.: The Cochlear Nerve and the Vascular Theory of Nerve Deafness, J. Laryng. & Otol. **55**:531 (Dec.) 1940.

48. Shambaugh, G. E., Jr.: Diplacusis: A Localizing Symptom of Disease of the Organ of Corti; Theoretic Considerations, Clinical Observations and Practical Application, Arch. Otolaryng. **31**:160 (Jan.) 1940.

On theoretic grounds the author believes that displacusis can be caused only by a distortion of the vibrating membranes of the organ of Corti. In a series of 45 patients with diplacusis there was evidence of an inner ear lesion in all except 1 in whom the hearing was normal in both ears. The conditions in which diplacusis was found were: acute suppurative otitis media with serous labyrinthitis, chronic suppurative otitis media with serous labyrinthitis, otosclerosis of the labyrinthine type, serous labyrinthitis following fenestration of the labyrinth, probable hemorrhage into the labyrinth, acute acoustic trauma to the labyrinth, labyrinthitis secondary to focal infection and labyrinthitis due to a food allergy.

The author concludes that diplacusis means a lesion of the membranous labyrinth in the cochlea, with distortion of the vibrating membrane of the organ of Corti and that as such it is of great value in localizing the pathologic changes in cases of nerve deafness.

With respect to nerve deafness due to central lesions, Fischer [49] studied the temporal bones from 5 patients in whom brain tumor had not been treated surgically and there was evidence of increased endocranial pressure. The histologic findings were similar to those observed by the same author in 5 previous cases of brain tumor and were as follows:

1. Transudation, exudation and lymph congestion in the region of the membranous labyrinth and nerve ganglion apparatus.

2. Hyperemia, hemorrhage and changes in the blood vessels of the membranous inner ear and petrous bone, the result of venous congestion.

3. Ectasia, stenosis or collapse of the ductus cochlearis.

4. Hernias of the brain on the anterior surface of the pyramid.

5. Late changes, consisting of pigmentations, adhesions and atrophy in the membranous labyrinth and organ of Corti.

6. Atrophy of the bone of the pyramid due to brain pressure.

The pathways by which brain pressure is transmitted to the inner ear are the subarachnoid spaces of the aquaeductus cochleae, the nerve sheaths of the internal auditory meatus, the pacchionian granulations, the spiral ligament and the lymph channels and blood vessels of the bony labyrinth.

The symptoms of congestion of the inner ear are tinnitus, decrease in bone conduction and loss of hearing beginning in the high tones. Congestive changes in the inner ear may be present for a time without these symptoms, just as choked disk may occur without loss of vision

49. Fischer, J.: Changes in the Internal Ear Due to Increased Endocranial Pressure, Arch. Otolaryng. **32**:391 (March) 1940.

for a time. Long-standing congestion of the inner ear results in secondary degenerative processes beginning at the basis of the cochlea and extending toward the apex. Prevention of these irreparable changes depends on early surgical intervention to relieve the increased endocranial pressure.

[Comment: That there is a condition in the ear analogous to choking of the disk has not been generally accepted. Brunner (see the review for 1937) stated that a tumor of the posterior fossa, especially of the cerebellopontile angle, can compress the veins from the inner ear, with stasis and accumulation of transudate in the spaces of this inner ear. This condition he called "stasis-hydrops of the inner ear." He did not regard it as comparable to choking of the disk, since it does not occur with general increase of intracranial pressure but only as a result of local compression of the venous return. Brunner and Fischer apparently are speaking of the same condition. They differ in their conception of its pathogenesis.]

Winston [50] states that the accepted syndrome for the diagnosis of a lesion of the cerebellopontile angle is total loss of function on the affected side, with loss of function of the vertical canals but retention of function of the horizontal canal and cochlea on the unaffected side. This typical syndrome is usually regarded as indicating a probable tumor of the acoustic nerve, whereas variations in this picture probably indicate a tumor in the cerebellopontile angle, involving the nerve secondarily.

The author analyzed a series of 34 cases of tumor of the cerebellopontile angle and found that only 57.8 per cent of the patients with acoustic nerve tumor showed the typical syndrome, while 62.5 per cent of those with extraneural angle tumors showed this syndrome. The author suggests that the term "cerebellopontile angle lesion syndrome" should be changed to "evidence of a peripheral lesion on the same side as the cerebellopontile angle lesion in addition to evidence of a central lesion," this evidence of a central lesion not being confined to loss of function of the opposite vertical canals but including: (1) spontaneous nystagmus on looking up, down or straight ahead; (2) disproportion between the durations of nystagmus and vertigo after rotation; (3) loss of function of the vertical canals or of the vertical and horizontal canals with good hearing on the opposite side, and (4) tolerance to vestibular stimulation.

With this new definition, the symptoms of 85 per cent of the patients with tumor of the cerebellopontile angle would conform to this syndrome.

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50. Winston, J.: A Revision of the Cerebellopontile Angle Lesion Syndrome, with an Analysis of the Vestibular Findings in Thirty-Four Cases of Verified Tumor of the Cerebello-Pontile Angle, Arch. Otolaryng. **32**:877 (Nov.) 1940.

In conclusion, the author states that he does not believe that one is justified in foretelling from a vestibular examination alone whether the anomaly is a primary or a secondary lesion of the angle.

[Comment: This is a valuable contribution based on a careful analysis of 34 confirmed cases of tumor of the cerebellopontile angle].

Brunner [51] states that histologically concussion of the brain consists of petechial hemorrhages with foci of crushing and degeneration in various parts of the brain stem and spinal cord. Clinically, after concussion of the brain there are headache, dizziness, tinnitus, nausea, emotional instability, depression and irritability, poor memory and poor concentration. This postconcussion syndrome may occur without loss of consciousness at the time of injury. The chief otologic manifestation of concussion of the brain is vertigo, usually without impairment of hearing or tinnitus. The vertigo may be labyrinthine, due to disease of the labyrinth or of the central vestibular pathways, in which case it is identical with the vertigo induced by caloric or rotation tests and is found associated with spontaneous nystagmus in the majority of cases if the patient is examined repeatedly and if the head is inclined backward. The labyrinthine tests usually reveal hyperexcitability, with increased nystagmus, but the results of the tests may be normal. The vertigo may be nonlabyrinthine, in which case it probably has nothing to do with the labyrinth or with the central vestibular pathways.

Fracture of the temporal bone complicates the picture of concussion of the brain. In transverse fractures the hearing and vestibular function are completely destroyed in nearly every case, while in longitudinal fractures the deafness is partial in the majority of cases, while vestibular function rarely remains.

In all cases of acute or chronic suppurative otitis media with transverse fracture a mastoid operation is indicated, to prevent intracranial extension. With longitudinal fracture and acute or chronic otitis the same indications for surgical intervention hold as without a fracture.

Concussion of the ear may complicate concussion of the brain. The pathologic changes of concussion of the inner ear are obscure, but probably there is a vascular lesion or a mechanical injury of the organ of Corti that was caused by a sudden strong fluid wave in the perilymph and endolymph. The chief symptom of concussion of the inner ear is deafness, usually unilateral and tending to increase as time goes on, whereas, with longitudinal fractures the deafness tends to abate.

[Comment: This is a somewhat dogmatic but therefore helpful summary of the aural lesions following trauma to the head.]

51. Brunner, H.: Disturbances of the Function of the Ear After Concussion of the Brain, Laryngoscope **50**:921 (Oct.) 1940.

Ménière's Syndrome.—Simonton [52] summarizes the results of treatment in 182 cases of Ménière's symptom complex at the Mayo Clinic. Medical treatment, consisting of a modification of the Furstenberg regimen, gave complete relief from vertigo in 34 per cent of the cases, partial relief in 28 per cent and no relief in 38 per cent. This treatment was more successful in patients with unilateral deafness and tinnitus than in those with bilateral deafness. Hearing was improved in 28 per cent and tinnitus in 33 per cent. In 35 cases complete relief from vertigo occurred six months to three years after the treatment was discontinued. For those patients not relieved by medical management total or subtotal intracranial section of the eighth nerve is the treatment of choice.

Lathrop [53] followed 68 patients with Ménière's symptom complex for periods of one to five years and found that 85 per cent were entirely satisfied with the results of the low sodium regimen advised by Furstenberg, Lashmet and Lathrop. Every patient should be given the benefit of adequate medical treatment before surgical intervention is considered. In the event that medical therapy fails, hemisection of the eighth cranial nerve should be the surgical procedure of choice.

[Comment: The use of section of the eighth nerve only after medical treatment has been given a trial and has failed seems a more reasonable procedure than that advocated by Dandy and the other members of the Johns Hopkins group, who consider section of the eighth nerve the treatment of choice in all cases.]

Biancalana [54] achieved complete relief from dizziness in 3 cases of Ménière's disease by section of the vestibular nerve. In 2 of these Ménière's disease was preceded by otitis. The author advises emptying the cisterna magna and the cisterna sulci lateralis, in order to move the cerebellum and gain access to the nerve. He notes that the anterior part of the nerve is the vestibular portion and that transient facial paralysis is a frequent occurrence after the operation.

[Comment: Dizziness complicating acute or chronic otitis is usually the result of perilabyrinthitis with secondary serous labyrinthitis, and treatment should be directed toward eradication of the causative bone disease rather than toward section of the eighth nerve.]

Kawata [55] describes a patient with attacks of dizziness and tinnitus following trauma to the left occiput. The hearing in the left ear was

52. Simonton, K. M.: Ménière's Symptom Complex: A Revision of the Literature, Ann. Otol., Rhin. & Laryng. **49**:82 (March 1940).

53. Lathrop, F. D.: Treatment of Ménière's Syndrome Complex, S. Clin. North America **20**:721 (June) 1940.

54. Biancalana, L.: Resezione parziale dell'austico per sindrome di Ménière, Boll. e mem. Soc. piemont. de chir. **9**:508, 1939.

55. Kawata, S.: Ménièrescher Symptomenkomplex nach Trauma, Oto-rhinolaryng. **12**:708, 1939.

reduced. The author believes that his case is one of commotion of the labyrinth.

[Comment: This case appears to fall in the group of "concussion of the ear" in Brunner's [51] classification of vertigo following trauma.]

Güttich [56] emphasizes the value of positional nystagmus in the differential diagnosis of vertigo from the cerebellum or from the labyrinth: In cerebellar disease the direction of the nystagmus varies with the position of the head, whereas in extracerebellar disease the nystagmus does not change with change of position.

Van Caneghem [57] studied the cerebrospinal fluid pressure in 30 cases of Ménière's disease. While the pressure was elevated somewhat in all cases, it was still within the normal range in more than half, and high pressure was observed in only a small group of patients, usually in those with neurovascular disturbances, extradural inflammation or trauma. Raising the spinal fluid pressure with the administration of amyl nitrite in no case produced an attack of vertigo. In 1 case the spinal fluid pressure was measured before and after an attack and was not found to be raised. However, in 2 cases it was possible to terminate the attack of vertigo by administration of amyl nitrite, and in 1 of these the pressure was doubled by this medication. As a result of his observations the author believes that Ménière's disease is not due to a disturbance in the circulation of the cerebrospinal fluid.

Talbott and Brown,[58] in 48 cases of Ménière's syndrome, made determinations of the acid-base constituents of the blood, including total fixed base, sodium, potassium, calcium, chloride, total carbon dioxide, phosphate, protein and nonprotein nitrogen. No constant variation in these constituents was found between attacks. In 4 cases studied during an attack, however, an increased concentration of serum potassium and a decreased concentration of serum sodium were noted.

As a working hypothesis the authors suggest that an increase in serum potassium indicates a loss of tissue potassium, just as a high level of serum calcium accompanies loss of calcium from the tissues. By administration of additional potassium the depleted tissue potassium is restored and equilibrium established. From 6 to 10 Gm. of potassium chloride in aqueous solution was given daily, without other dietary modification. Forty patients maintained for one to sixteen months on this dosage were all benefited, although none was cured.

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56. Güttich, A.: Ueber den Ménièreschen Symptomenkomplex und seine Behandlung, Therap. d. Gegenw. **81**:292 (Aug.) 1940.

57. van Caneghem, D.: La pression du liquide cérébro-spinale dans la maladie de Ménière, Ann. d'oto-laryng, January-February 1940, p. 36.

58. Talbott, J. H., and Brown, M. R.: Ménière's Syndrome: Acid-Base Constituents of the Blood; Treatment with Potassium Chloride, J. A. M. A. **114**: 125 (Jan. 13) 1940.

The authors suggest that the good results previously reported with a low sodium diet were due to a relative increase in the intake of potassium as compared with that of sodium. Seven patients previously treated with a low sodium diet without improvement all improved under the higher potassium regimen. The authors found that a return of symptoms was noted in most patients who did not follow directions and that a return to the regimen was accompanied by prompt alleviation of symptoms. Surgical section of the eighth nerve has not been resorted to in any case since the institution of this new treatment.

Walsh and Adson [59] reviewed 186 cases of Ménière's syndrome in which treatment was given at the Mayo Clinic and found that in 62 per cent of the cases the vertigo was alleviated or made to disappear by the use of a low salt diet alone or by administration of ammonium chloride or potassium nitrate. Of 23 patients treated by other general measures, including elimination of foci of infection, relief or improvement occurred in 78 per cent, but in this group the improvement did not immediately follow treatment, whereas in those treated by diet and administration of ammonium chloride or potassium nitrate the improvement occurred promptly. Surgical treatment was carried out on 22 patients, 9 of whom had been placed on medical treatment with only partial or temporary or no improvement. Of 13 patients on whom complete section of the eighth nerve was carried out, 9 obtained complete relief from their vertigo, 3 obtained great improvement and 1, no improvement. Of 7 patients on whom partial section of the nerve was performed, 4 were completely relieved but lost more or all of their hearing, while 3 who retained their hearing were greatly improved.

[Comment: Although the authors do not appear to regard removal of foci of infection as of value in Ménière's syndrome, their statistics indicate that "general measures with removal of foci" resulted in better eventual results than medical treatment. The results of surgical section reported by Adson are in contrast to Dandy's dogmatic statement that vertigo is always cured by section of the nerve.]

Wright [60] treated 84 consecutive patients with Ménière's disease by removal of foci of infection in the teeth, tonsils and sinuses. In 68, the tonsils were found to be infected, in 27, the teeth and in 20 the sinuses. No other treatment was given. Six months after treatment it was found that 70 of the 84 patients were cured of their vertigo, 4 were improved, 6 were unchanged and 4 were worse; 2 were cured of their deafness, 46

59. Walsh, M. N., and Adson, A. W.: Ménière's Syndrome: Medical Vs. Surgical Treatment, J. A. M. A. **114**:130 (Jan. 13) 1940.

60. Wright, A. S.: Further Clinical Observation on the Nature and Treatment of Ménière's Disease, J. Laryng. & Otol. **55**:388 (Aug.) 1940.

improved, 14 unchanged and 6 worse; 9 were cured of their tinnitus, 25 improved, 17 unchanged and 6 worse.

The author notes that some of the improvements might have occurred in the normal course of the disease, but since the average duration of the disease in this series was six years, the results must be attributed to the treatment. Of the 10 patients in whom the treatment resulted in failure—i. e., those whose vertigo was worse or unchanged, 7 had chronic sinusitis and 8 had multiple foci. Three of these have pronounced bacterial allergy, 1, generalized edema, 1, sciatica and 1, chronic progressive choroiditis. The author suggests that the failure of others to duplicate his results may be attributed to lack of thoroughness in the removal of foci, due to halfhearted acceptance by these physicians of the idea of an infectious etiologic agent.

[Comment: The author's description of his 10 failures suggests that underlying allergy might have been responsible for many or most of them. His results compare favorably with those reported following dietary treatment. My own experience supports the author's contention that focal infection is one of the frequent causes of Ménière's syndrome.]

Meyer [61] reports 12 cases of allergic irritation of the inner ear with the symptoms of Ménière's disease. He believes that in obscure cases of Ménière's disease one should consider the possibility of allergy but should not rely on skin tests alone.

Atkinson [62] reports the case of a man aged 32 with deafness, tinnitus and mild dizziness, "bilious" attacks, stuffy nose, postnasal drip and recurring redness of the eyes. There were no characteristic symptoms of allergy, but because he drank about 2 quarts of milk daily, milk was removed from his diet, with almost miraculous results. Ingestion of a cheese sandwich resulted in a return of all his symptoms, confirming the allergy to milk.

In the treatment of Ménière's disease the author recommends the removal of foci of infection, treatment of allergic conditions and elimination of the use of alcohol and tobacco. He has had no success with the salt-free diet or with ammonium chloride or potassium chloride. Section of the eighth nerve has a real but limited application and should be employed only after thoroughly conservative measures have failed. In conclusion, the author states that the breadth of vision of the general physician and not the keyhole view of the specialist is needed in Ménière's disease.

[Comment: Since Ménière's syndrome is a disease of the inner ear, it falls within the domain of the otologist. The conscientious and well trained specialist will resent the author's use of the term "keyhole view

61. Meyer, C.: Ueber allergisch bedingten Reizungen des inneren Ohres, Pract. oto-rhino-laryng. **3**:92, 1940.

62. Atkinson, M.: Vertigo, Canad. M. A. J. **42**:326 (April) 1940.

of the specialist." In my experience the competent otologist is in a better position than the general physician to evaluate the symptoms of dizziness, to differentiate accurately true aural vertigo from dizziness due to other causes, such as cerebral vascular instability, to discover and treat vertigo due to chronic perilabyrinthine osteitis with chronic suppurative otitis media and to determine whether removal of foci, removal of allergic factors, dietary management or section of the nerve is required. The otologist, more than the general physician, has been responsible for such recent advances in the knowledge of the pathologic changes, the causes and the treatment of Ménière's disease as the histologic studies of ears (Hallpike), the conception of an allergic cause in certain cases of Ménière's disease (Dean, Jones, Dohlman and others), the dietary treatment (Furstenberg) and the removal of foci of infection (Wright).

The last three years have seen a great awakening of interest in Ménière's syndrome. For the first time since Ménière's first incomplete pathologic report there have been careful histologic studies of the ears, showing in every case evidence of a marked increase in pressure of the endolymph. In addition, I have found and reported diplacusis in nearly all the patients with Ménière's syndrome examined by me, indicating a distortion of the organ of Corti, presumably as a result of edema of the vibrating membranes. Serous labyrinthitis, the result of focal infection, could produce such edema and increase of endolymph pressure. Allergic irritation of the labyrinth might also cause edema and increase of endolymph pressure. Recent observations by Dean, Jones, Dohlman, Shambaugh Jr. and others indicate that allergy, usually to a food, is a not uncommon cause of Ménière's syndrome, while observations made by Wright, Shambaugh Jr. and others indicate that focal infection is an important cause of the condition.

The complete picture of Ménière's syndrome is gradually taking form as follows:

In the majority of cases the disease begins as serous labyrinthitis from focal infection or as allergic labyrinthitis from a food allergy, but in a few cases chronic suppurative otitis media or a vascular anomaly pressing on the eighth nerve is the cause.

Serous or allergic labyrinthitis results in increase of endolymph pressure and irritation of the labyrinth with dizziness and tinnitus, usually with depression of function, cochlear or vestibular or both.

Lowering of the increased endolymph pressure by decrease of the fluid intake (Mygind and Dederding), the use of a low salt diet (Furstenberg), the administration of ammonium chloride, potassium chloride or potassium nitrate (Furstenberg, Talbot and Brown and Walsh), may alleviate the symptoms. However, it would appear more logical to search for and remove the etiologic factors, which in most cases will be foci of

infection or an allergic substance, usually a food, and to employ the dietary measures when no etiologic factor can be found or when its removal does not result in prompt relief. Section of the eighth nerve must be regarded as an admission of defeat in the effort to determine the cause and should become less frequent as methods of diagnosis and treatment, particularly of food allergy, improve. At the present time detection of the allergic cases is most difficult, because in the majority of food allergies the skin tests are unreliable, and as changes in the diet are made, new food allergies may develop. One must therefore rely on various forms of elimination diets or on "hunches"—such as the elimination of milk because a patient drinks a large amount of it.

Finally, it is now clear that the serous or the allergic labyrinthitis frequently responsible for Ménière's syndrome tends to run a self-limited course, so that after a varying period the attacks of dizziness disappear or are greatly abated, the progressive loss of hearing halts and the tinnitus lessens. The same tendency to recovery is seen in other conditions that are the result of focal infection and allergy, and this tendency must be taken into account in evaluating any form of treatment.

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Abstracts from Current Literature

Ear

War Deafness and Its Prevention. Stacy R. Guild, Ann. Otol., Rhin. & Laryng. **50**:70 (March) 1941.

War deafness, as usually understood, is the deafness produced by acoustic trauma due to exposure to recurrent extremely loud noise for long periods and detonation waves from nearby explosions. During war, however, the incidence of hearing impairment from a number of causes is far greater in the military personnel than in the corresponding age group in time of peace. Epidemic cerebrospinal meningitis with complicating labyrinthitis, otitis media with the various complications, direct injury by wounds and rapid changes of altitude in combat flying are large factors in this increase, in addition to the acoustic trauma. Control of meningitis and its complications is greatly aided by chemotherapy. All measures possible should be taken to reduce the incidence and severity of infections of the upper respiratory tract. Guild pleads for a better physical examination of candidates, to weed out all those who have incipient pathologic conditions affecting the ears, as many of these will eventually have to be discharged, and thus prevent economic loss in wasted training, hospital care and eventual pensions. Steel helmets reduce the incidence of direct injury by wounds. Prevention of injury due to rapid changes in atmospheric pressure while flying has not been satisfactorily worked out.

Acoustic trauma causes more impairment for high tones than for low, those above 2048 or 4896 cycles being involved and reception for conversation being largely retained. The lesion is probably in the basal turn of the cochlea and consists of more or less destruction of the organ of Corti and atrophy of nerve fibers. No known treatment is effective. A single detonation may cause severe and permanent impairment even for reception of loud conversation. Frequently, the drum membrane is ruptured by the force of a blast. Unless infection occurs, the drum membrane frequently heals, but attempts to clean the external canal often set up infection. Animal experiments show that it is probable that there is in man extensive damage to the cochlea and nerve. Experiments also seem to indicate that rupture of the drum membrane tends to protect the inner ear, cochlear lesions being more severe and extensive in the cases in which the drum was not ruptured than in those in which it was.

To prevent acoustic trauma, the intensity of the sound or detonation waves has only to be reduced sufficiently, and not necessarily stopped entirely. Most methods of gaining this end have been unsatisfactory. Solid obturators, if properly fitted, reduce the waves, but the constant irritation of wearing tends to cause infection. Cotton plugs soaked in an ointment also protect well, but both these methods are unsatisfactory if the wearer has to listen for orders. Various types of obturators have been developed which work well under laboratory tests but which have hazards in military life in that if struck by a missile they shatter and produce large wounds. Guild discusses one made by a British firm during the last war, the "scientific ear drum protector 'Tommy'," which consists of a hollow spherical soft rubber bulb with an opening in one side and the short-necked opening surrounded by a soft rubber flange. This proved satisfactory for sailors in the United States Navy, near the muzzle of large caliber guns.

M. V. Miller, Philadelphia.

TINNITUS AURIUM IN THE LIGHT OF RECENT RESEARCH. E. P. FOWLER, Ann. Otol., Rhin. & Laryng. **50**:139 (March) 1941.

Fowler discusses his own observations and interpretations in the study of tinnitus. He divides tinnitus into two fundamental types: (1) Vibratory, which is mechanical in origin and caused by a to and fro response of the mediums in the ear to vibrations which may arise without or within the ear or be brought to the ear through the solid liquid or gaseous mediums of the body. The common causes are muscle spasm, vascular bruits from venous or arterial channels and nasopharyngeal squeaks, clickings, droppings and similar phenomena. (2) Non-vibratory, which is due to biochemical irritation of the neural mechanism and is an illusion of sound. These may develop through pressure; degenerative or regenerative lesions; anoxic, toxic or traumatic lesions, and, indirectly, by disorders of the endocrine and autonomic nervous systems. The two types of tinnitus may occur separately or together.

The "loudness balance" test is discussed—that is, balancing the tinnitus with the sound being used for comparison. Fowler found that in attempting to measure tinnitus a 20 decibel increase of the sound in a nerve-deafened ear appears like a greater than 20 decibel increase, often appearing as more than a 40 or even more than a 60 decibel increase, as compared with the increase perceived by an opposite normal or obstructively deafened ear. He also found that the rate of increase in loudness accelerates rapidly with intensity, so that for very loud sounds there is often little or no difference in the sensation of loudness when the same sound is applied to a normal or a nerve-deafened ear. The increase of loudness with intensity he calls the "recruitment phenomenon." He believes that it is a positive and reliable sign for diagnosing nerve deafness.

The association of tinnitus with deafness, the factor of fatigue, the mechanism of bilateral tinnitus of peripheral and central origin and neurophysiologic, pathologic and other elements in connection with tinnitus are discussed.

M. V. MILLER, Philadelphia.

LABYRINTHITIS FROM LIGHTNING. L. CHAVANNE, Oto-rhino-laryng. internat. **25**:17 (Feb.) 1941.

Two observations are reported by Chavanne. The first concerns a man aged 48 years who after a stroke of lightning in his vicinity complained of vertigo, diminished hearing and tinnitus in the right ear. Diminished sensation on the side of the face, the cornea, the neck and the arm on the right side, lateropulsion and deviation of the left arm to the right and diminished sensibility of the right labyrinth were observed. Vasomotor disturbances of the right side of the face were marked. There was spontaneous horizontal rotatory nystagmus to the left, but the vertigo was not organized and did not occur in crises. Probably the cause of the condition was a cerebral vasomotor disturbance with a slight labyrinthine involvement.

The second case was that of a man aged 40 years who, after a stroke of lightning, suffered crises of vertigo with vomiting for a week, accompanied by marked deafness. The hearing quickly improved, but the vertigo and headaches continued. There was no spontaneous nystagmus or deviation. A caloric test revealed hypersensitivity of both vestibular organs. The case is regarded as one of typical vertigo-neuralgia.

DENNIS, San Diego, Calif.

RELATION BETWEEN THE PNEUMATIZATION OF THE TEMPORAL BONE AND COMPLICATIONS OF ACUTE MIDDLE EAR INFECTION. HELMUTH RICHTER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:421 (June) 1940.

Richter analyzed his operative cases of mastoiditis of the past eleven years, in which various forms of intracranial complications have occurred. The 213 patients studied presented a total of 260 complications of all types. He classified the pneu-

matization of the temporal bone as (1) good, (2) moderate or (3) very poor. While many of the benign complications, such as extradural abscess in the middle or the posterior fossa and Bezold's abscess, occur frequently in temporal bones with extensive pneumatization, he found that the more serious complications usually occur when the pneumatization has been limited.

He believes that a temporal bone with poor pneumatization deserves a considerable amount of watchfulness. Such a temporal bone influences the course of each acute middle ear infection and acts as an indicator for mastoidectomy; it also adds another important factor in the possibility of the development of many intracranial complications which are dangerous to life.

PERSKY, Philadelphia.

PRIMARY AND SECONDARY TUMORS OF TEMPORAL BONES. WERNAR FEHRE, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:442 (June) 1940.

Fehre reports in detail a series of tumors of temporal bones. There were 1 primary and 7 secondary tumors. These included myeloma, neuroma, epithelioma and carcinoma. A secondary tumor of the temporal bone may be metastatic from a bronchus, the stomach, the meninges or any other organ.

PERSKY, Philadelphia.

SEASONAL VARIATIONS INFLUENCING OTITIS MEDIA. K. SCHEFFLER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **47**:32 (Aug.) 1940.

Scheffler undertook a study of climatic and seasonal influences on otitis media. He found that pneumococcic infection was the only type of infection that was frankly influenced by seasonal variations. The influence of meteorologic factors was negligible. In studying the influences of air currents from various sections of the country, he found that in the maritime subtropical regions, wherein there is a marked and rapid variation between the extremes of hot and cold, there was a definite increase in otitis media in general. But where there was more or less constant arctic weather, the incidence of otitis media was comparatively rare.

PERSKY, Philadelphia.

COMMENTARY ON THE FACTORS WHICH MAY DEFEAT THE TREATMENT OF OTITIC MENINGITIS WITH SULFANILAMIDE AND ITS DERIVATIVES. G. BUZOÏANO, Acta oto-laryng. **29**:113, 1941.

If the two principles in the care of otitic meningitis have been carefully realized and executed, namely, (*a*) complete surgical exenteration of the septic focus in the middle ear and mastoid by radical mastoidectomy and (*b*) administration of massive doses of the indicated chemotherapeutic agent (depending on the organism encountered) and if the cure of the meningitis is retarded or fails, one must take into consideration the following factors which can operate to prevent recovery:

1. Impermeability of the meninges to sulfanilamide and its derivatives.

2. Presence of an undiscovered intracranial focus of infection which continues to feed the subarachnoid spaces.

3. Otitic meningitis of labyrinthine origin. If this is present, it is necessary to open the labyrinth at all costs.

4. Presence of meningitis due to a mixture of organisms, both bacillary and pyogenic.

5. A septic ventricular block insufficiently controlled by chemotherapy, either oral or intrathecal.

Failure may occur also in cases in which the meningitis is in a late stage and the fluid very purulent, in cases in which the patient is seen very late in the course of the disease and in cases of meningitis associated with thrombophlebitis.

GROVE, Milwaukee.

Pharynx

ANGINA OF PNEUMOCOCCIC ORIGIN. ARISTIDE MONTEIRO, Brasil-med. **51**:193 (Jan.) 1937.

Monteiro encountered a group of cases in which acute angina did not respond to bismuth therapy. In analyzing these cases, he found that the causative organism was Pneumococcus

He reports in detail 3 cases in which a change of therapeusis was necessitated because of pneumococcic infection. He then summarizes the literature, outlining the symptoms, diagnosis and genesis of pneumococcic angina.

He states that the prognosis is usually benign but that the course is of long duration. Frequently when the infection is due to a mixture of bacteria, septicemia is a complication. This naturally affects the prognosis and makes the ultimate result uncertain. Associated localized cervical adenitis is usually of secondary importance. It depends on the intensity of the local reaction and, as a rule, has no effect on the final prognosis. While a number of deaths have been reported in the literature, Monteiro believes that the favorable climate of Brazil has a marked influence on pneumonia in general and particularly on the local manifestation in the throat. It is known that in Brazil pneumococcic infections usually run a very mild course, more or less benign in character, and that the prognosis is usually favorable.

Monteiro then discusses treatment of pneumococcic angina. He employs local treatment to the throat in the form of alkaline gargles and applications of mild disinfectants. On the other hand, he stresses the value of both serotherapy and vaccinotherapy and of employing the specific type of serum or vaccine for the particular type of Pneumococcus which is present. He also states that in some cases the condition may be favorably influenced by a polyvalent serum. He administers either 50 cc. of the specific serum or five hundred million germs of the vaccine. While usually one injection is sufficient, occasionally there may be need for repetition. (No mention has been made of the use of sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) in this form of infection. It seems that this should be included at present in the armamentarium for treatment of every form of pneumococcic infection.)

PERSKY, Philadelphia.

TONSILLECTOMY IN CASES OF PERITONSILLAR ABSCESS. W. HAARDT, Monatschr. f. Ohrenh. **75**:93 (Feb.) 1941.

In 100 cases of peritonsillar abscess, five types of tonsillectomy were used: unilateral, with the patient under general anesthesia; bilateral, with the patient under general anesthesia; unilateral, with the region under local anesthesia; bilateral, with the region under local anesthesia, and tonsillectomy during the "lukewarm" stage. Ninety-four of the patients recovered without any complications, 2 died "without death being attributable to surgery" and 4 recovered despite complications (sepsis). According to Haardt, a review of the literature reveals excellent results in thousands of cases. The best and simplest results are achieved with unilateral tonsillectomy with the patient under a light general anesthesia. In bilateral tonsillectomy the uninvolved side often bleeds more than the abscessed side. In several cases in which the operation was performed with the region under local anesthesia, a slight increase in temperature was noted after operation.

In general it must be admitted that 87 to 99 per cent of all peritonsillar abscesses will heal either by spontaneous rupture or after incision. However, in cases of deep abscess, insufficient drainage, phlegmon, nephritis, sepsis and the like, "abscess-tonsillectomy" should be performed, combined eventually with an external operation. Tonsillectomy in the "lukewarm stage," two to seven days after incision of the abscess, has given rise to septic conditions and therefore has been discontinued. A symptom which might be useful in diagnosing hidden abscess consists of provoking a retching reflex. Normally the tonsil and posterior pillar

move toward the midline. This is lacking in the presence of an abscess. Seventy-four per cent of the abscesses were found near the upper pole, 17 per cent next to and back of the tonsil and 6 per cent (small abscesses) close to the inferior pole.

LEDERER, Chicago.

TREATMENT OF FOLLICULAR FOCAL INFECTION FROM THE STANDPOINT OF THE INTERNIST, WITH SPECIAL REFERENCE TO ELECTROCOAGULATION OF TONSILS. MAX E. BIRCHER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **47**:14 (Aug.) 1940.

Bircher discussed the various methods of treatment of infected tonsils. These methods include: (1) gargling; (2) local application of drugs, such as mild protein silver, to the tonsils; (3) insufflation of various powders; (4) suction of infected material from the tonsils; (5) irrigation of the crypts of tonsils with various solutions; (6) incision of the tonsils, and (7) excision.

The author outlined the limitations and contraindications of each method. His choice of methods is the electrocoagulation of tonsils. He suggested the following indications: old age, rheumatic fever, hemophilia, acute tonsillitis, chronic infections in orators and singers, when the time element is a factor, and finally, fear of the operative procedure on the part of the patient.

The author then describes in detail his method of treatment employing the unipolar electrode and presents reports of a series of favorable results, including photographs which show the pathologic changes resultant from electrocoagulation.

PERSKY, Philadelphia.

Larynx

HAEMOPHILUS INFLUENZAE TYPE B IN ACUTE LARYNGITIS WITH BACTEREMIA. SYDNEY E. SINCLAIR and MILDRED D. FOUSEK, J. A. M. A. **117**:170 (July 19) 1941.

Sinclair and his co-worker studied in the clinic 8 patients with acute laryngitis and bacteremia due to Haemophilus influenzae type B. They speculated as to whether or not there may be a relationship between influenza of this type and that of the epidemic type prevalent in the United States in 1940-1941.

A review of the cases of these patients, together with 2 encountered in previous years, reveals a constant clinical picture of severe prostration, in addition to the obstructive symptoms of the larynx. In 6 cases the obstructive symptoms were severe enough to warrant tracheotomy, and in 2 cases the latter procedure was insufficient, for the patients died.

The following observations are offered:

1. H. influenzae type B was recovered from cultures of the blood of 10 patients with acute laryngitis.

2. The occurrence of H. influenzae type B bacteremia in cases of acute laryngitis produces a septic syndrome adding to the mechanical hazard of that disease.

3. The presence of this organism in the nasopharynx may be detected within a few hours by means of a nasal swab culture.

4. The use of drugs of the sulfonamide series has proved a valuable means of treatment in addition to the relief of mechanical obstruction.

5. The use of H. influenzae type B rabbit antiserum is suggested for patients not responding to tracheotomy and chemotherapy.

GORDON, Philadelphia.

PARALYSIS OF THE RECURRENT LARYNGEAL NERVE IN HUMAN BEINGS. GUSTAV HOFER and JOSEF JESCHEK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:401 (June) 1940.

Hofer and Jeschek discuss in detail the conclusions reached by many investigators concerning the physiopathology of paralysis of the recurrent laryngeal nerve. They believe that there is a fallacy in most of the current thought con-

cerning the causes and the method of production of various types of paralysis of the larynx. In fact, they believe that Semon's law concerning the position of the vocal cords following either an injury or the cutting of the recurrent laryngeal nerve is not infallible. They object to the use of animal experimentation as a means of deducing the physiopathologic results of injury to the recurrent nerve. Anatomically, there is a difference between experimental animals and man.

Hofer and Jeschek reported a series of cases in which there were various types of lesions in and about the larynx. In these the vagus nerve, the recurrent laryngeal nerve or the internal or the external branch of the superior laryngeal nerve was either cut or injured. As a result of their observations in these cases, they drew the following conclusions: 1. Severance of the recurrent laryngeal nerve produces paralysis of the vocal cords in a (para) median position. 2. High severance of the vagus nerve gives an intermediate or cadaveric position of the vocal cords. 3. The cutting of the internal branch of the superior laryngeal nerve does not influence the position of the cord, which proves that it does not supply any motor fibers to the larynx. 4. Cutting of the external branch of the superior laryngeal nerve, because of its action on the cricothyroideus muscle, has a definite influence on the position of the cord. Bilateral cutting of these nerves causes a triangular opening of the cords. 5. Cutting of the recurrent laryngeal nerve and the internal branch of the superior laryngeal nerve has the same effect as cutting of the recurrent nerve. 6. Cutting of both the recurrent laryngeal and the external branch of the superior laryngeal nerve gives the typical picture of so-called recurrent paralysis—that is, an intermediate position, as evidenced by abduction of the cords, and a slight internal rotation and a slight tilting internally of the arytenoid cartilage.

Although the authors have made some deductions as to the motor innervation of the larynx, the importance of their observations rests on the fact that although injury to the recurrent nerve produces a median position of the vocal cords, it does not necessarily mean that there is a disturbance of speech. Care should be exercised whenever one operates in the vicinity of the external branch of the superior laryngeal nerve, as injury to this nerve definitely affects speech. Finally, the authors believe that all is not known concerning the motor innervation of the larynx and recommend further study.

PERSKY, Philadelphia.

GRIPPAL TRACHEOBRONCHITIS AND ITS TREATMENT. R. PERWITZSCHKY, Ztschr. f. Hals-, Nasen- u. Ohrenh. **47**:1 (Aug.) 1940.

Perwitzschky reports 5 cases of acute tracheobronchitis following grippal infection, which were characterized by marked difficulty in breathing, both in the inspiratory and expiratory phases. In each of his cases the characteristic stridor was definite. The upper respiratory tract was essentially normal, but when a laryngeal examination was possible, he found a pseudomembrane which covered the entire lining of the trachea and the bronchi. Although this pseudomembrane was easily removed, there was a marked tendency toward crust formation. This was noted particularly in the cases in which tracheotomy had been performed.

He discusses the pathologic and the bacteriologic aspects of these membranes and recommends as the ideal methods of treating this malady the administration of morphine and anastil (an expectorant containing guaiacol) and the use of steam inhalations and of warm oily sprays directed into the trachea.

The prognosis is usually grave, particularly for younger children. Tracheotomy usually favors the drying of the secretion with formation of crusts, which may occlude the entire lumen and produce a cast of either the trachea, the bronchi or both. Some of these casts may be so extensive as to occlude the lumens of these structures completely.

In conclusion, Perwitzschky states that the course of tracheobronchitis following grip is so characteristic that the disease must be differentiated from other forms of tracheitis and diphtheria.

PERSKY, Philadelphia.

A Case Report of an Infected Laryngocele. H. Videbech, Acta oto-laryng. **29**:123, 1941.

Videbech reports the case of a 62 year old man with an abscess of the neck which was repeatedly incised, with evacuation of pus, and as often recurred. The condition had been wrongly diagnosed. The diagnosis of infected laryngocele was then made. The growth was extirpated by external operation, and the patient was cured. Videbech calls attention to the scarcity of laryngocele in general (only 60 to 79 have been reported in the literature). Of these, about 10 per cent became infected.

Laryngoceles are divided into symptomatic and true varieties. The symptomatic variety arises on the basis of a chronic granuloma, such as that associated with syphilis or tuberculosis, which causes a partial closure of the ventricle of Morgagni. During phonation the ventricle becomes dilated with air and is enlarged upward or downward. The true laryngocele is probably congenital in origin and arises from an abnormal development of the sinus of Morgagni. The enlargement of the sinus of Morgagni takes place internally (laryngocele interna), or a herniation through the thyrohyoid membrane takes place (laryngocele externa), or the laryngocele may be of a combined internal and external type. Some of the factors which tend to enlarge a laryngocele are severe attacks of coughing, vomiting or crying and the use of wood wind and brass musical instruments.

Grove, Milwaukee.

Miscellaneous

Relation of Tonsillectomy and Adenoidectomy to the Incidence of Poliomyelitis. A. E. Fischer, M. Stillerman and H. H. Marks, Am. J. Dis. Child. **61**:305 (Feb.) 1941.

Because a high incidence of bulbar poliomyelitis was reported in patients who were recently tonsillectomized, a study was made of clinical cases to put this observation on a firmer basis. Among 507 patients who were treated for poliomyelitis in Toronto in 1937, there were 231 whose tonsils and adenoids had been removed. The date of operation was obtained for every tonsillectomy performed.

It was found that the proportion of cases of bulbar poliomyelitis was higher, and that of the nonparalytic form was lower, among those tonsillectomized within a month of the onset of the disease than among those operated on more than a month prior to the onset of the poliomyelitis. The percentage of cases of the bulbar form among the children who had been subjected to tonsillectomy, regardless of when this was performed, was over twice as high as that among nontonsillectomized children, 18.7 and 7.5 per cent, respectively. This ratio should probably be somewhat reduced, since patients with this form of disease cause greater concern and are therefore more frequently transferred to the hospital for care. The mechanism of the lowered resistance to this disease, which is the probable explanation for its higher incidence in tonsillectomized children, is not entirely clear. It is probably related to the portal of entry of the disease or the avenue of its spread.

Robert B. Lewy, Chicago.

Primary Pituitary Adenoma and the Syndrome of the Cavernous Sinus. Laurence M. Weinberger, Francis Heed Adler and Francis C. Grant, Arch. Ophth. **24**:1197 (Dec.) 1940.

Of the classic triad of signs, the chiasmal syndrome, constitutional dyspituitarism and roentgen evidence of hypophysial disease, the first-mentioned has played the largest role in the recognition of pituitary adenoma. Indeed, the preeminence of the visual component in the semiology of pituitary adenoma led Henderson, who recently published a critical review of Cushing's series of 338 cases, to declare:

"It is exceptional for a chromophobe adenoma to be diagnosed or even suspected before it is sufficiently large to implicate the visual pathways. . . . operation would hardly be justified before vision is affected."

The occurrence of paralysis of the ocular nerves as an early, or in some cases the first, evidence of pituitary adenoma seems to have been first pointed out by Foix. He described a case in which a pituitary tumor first evidenced itself by severe pain in the region of the ophthalmic division of the fifth nerve and was associated with rapidly progressive unilateral paralysis of the third nerve, followed in turn by paralysis of the fourth and sixth nerves.

This accompanying paralysis of the oculomotor nerves as a syndrome of primary pituitary adenoma seems to have been neglected. From 169 cases of verified pituitary adenoma, the authors were able to select 14 cases in which unusual neuro-ophthalmic pictures permitted division on the basis of a cavernous sinus syndrome into three subgroups: (1) those in which the disturbances referable to the ocular and trigeminal nerves comprised the exclusive neurologic picture; (2) those in which the symptoms referable to the ocular and trigeminal nerves dominated the clinical picture but in which there were some evidences of implication of the optic chiasm, and (3) those in which the disturbances referable to the ocular and trigeminal nerves were an important part of the clinical symptoms but in which there were unequivocal visual field defects indicating an intrasellar lesion.

The disturbances referable to the ocular and trigeminal nerves in these cases were accounted for by the implication of the cavernous sinus. It was shown that occasionally a pituitary adenoma grows laterally and that this mode of growth may produce the clinical picture of a lesion in the sphenoid fissure rather than the classic chiasmal syndrome.

Of special importance in the presentation was the analysis of the anatomic basis of the syndrome. The essential etiologic factors naturally require that the pituitary adenoma grow out of the sella turcica laterally.

SPAETH, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

AUTOGENOUS CARTILAGE GRAFTS: AN EXPERIMENTAL STUDY. FORREST YOUNG, Surgery **10**:7 (July) 1941.

Experimental studies carried on with dogs, consisting of the transplantation of autogenous rib cartilage with and without perichondrium, uniformly showed that the transplanted cartilage remains living and maintains its size and weight for as long as one and one-half years.

Finely chopped autogenous rib cartilage seeded over the fascia of the rectus muscle produced a solid sheet of cartilage-like material, which is seen microscopically as particles of cartilage fused together with fibrous tissue. Grossly, the mass had some of the characteristics of fibroelastic tissue.

HENRY A. MILLER, Philadelphia.

THE SURGICAL MANAGEMENT OF POSTRADIATION SCARS AND ULCERS. HERBERT CONWAY, Surgery **10**:64 (July) 1941.

Clinical examination of the scars produced by roentgen radiation shows them to be characteristically of firm consistency, depressed below the adjacent surface and demarcated abruptly from it or blending gradually into the areas of normal skin. Often there is subcutaneous atrophy. The scarred area may be uniformly white or may show areas of pigmentation. There may be clusters of telangiectases, visible as reddened wormlike areas in the background of white scar tissue. In the most central portion of the scarred area, at the point of maximal ischemia, a punched-out indolent ulcer may occur. Malignant change either in the hypertrophic skin or in the wall of the ulcer has been noted.

The surgical management of these lesions presents problems which call for a knowledge of and the application of practically all of the principles of plastic

surgery. This may vary from serial excision and skin grafting to the use of pedicled tube grafts to provide bulk and closure. Ten cases, with their surgical management, are outlined.

HENRY A. MILLER, Philadelphia.

TREATMENT OF TRAUMA OF SKIN AND SUBCUTANEOUS TISSUES. F. W. BANCROFT, Surg., Gynec. & Obst. **72**:318 (Feb.) 1941.

Many failures in the treatment of acute traumas of the skin and soft tissues are due to a lack of consideration of pathologic lesions in the subcutaneous fat and connective tissues. The success or failure of the general treatment of contused wounds of the skin depends largely on tissue tension. Hematomas should be carefully aspirated under sterile technic, and in severe cases relaxation incisions may be indicated.

One might summarize the problem of primary or secondary closure of lacerated wounds as follows: Primary closure may be attempted if the patient is healthy, the skin is clean, the instrument causing the damage is relatively clean and sharp, and the patient has been seen well within six hours after the injury.

In regard to avulsed wounds, Farmer stated that thrombosis of the venous and lymphatic vessels is produced by the trauma, while the arterial vessels, which are not so easily injured, continue to pump blood into the traumatized zone. When the pressure of the incoming blood overcomes the normal tissue pressure, necrosis occurs, as the smaller vessels start to thrombose. Excision of the entire avulsed skin, with removal of all adherent fat, and laying this flap back as a free skin graft will yield excellent results, whereas ordinary suturing down of the avulsed piece would probably result in necrosis and sloughing.

H. A. MILLER, Philadelphia.

PERSISTENCE OF FUNCTION OF SKIN GRAFTS THROUGH LONG PERIODS OF GROWTH. JAMES B. BROWN and F. MCDOWELL, Surg., Gynec. & Obst. **72**:848 (May) 1941.

Satisfactory late function of grafts requires (1) enough skin for free movement, (2) moderate looseness, (3) ability to withstand usual trauma and (4) development of normal sensation. To insure best late results, exposed raw surfaces should be grafted early. Thick split grafts of considerable area can be grafted successfully. If the grafts are cut carefully, the same donor site can be used for several crops of grafts. Homografts may be used for temporary relief, in order to build up the general condition of the patient, even though they disappear in a short time. If grafting is undertaken early, much crippling and invalidism is prevented. The late results are highly satisfactory, as both thick split and full thickness grafts seem to grow, or at least to stretch out, with growth of the body area and to permit normal movement if they have been successful from the start.

H. MILLER, Philadelphia.

BELL'S PALSY AND HERPES ZOSTER. JOHN D. SPILLANE, Brit. M. J. **1**:236 (Feb. 15) 1941.

It is generally believed that Bell's palsy is caused by exposure to a cold draft, but there are other predisposing factors. Spillane states that serologic evidence supports the view that zoster infection is a probable, though uncommon, cause of Bell's palsy. He briefly describes an epidemic of herpes zoster during which 5 of the patients were placed in one ward at the same time. One case of combined trigeminal and geniculate zoster was encountered. Five days after facial herpes appeared ipsilateral facial palsy was noted. This suggests that the herpes virus invaded both the gasserian and the geniculate ganglion. That no auricular or faucial herpes developed indicates that herpetic invasion of the geniculate ganglion can occur without cutaneous manifestation in the distribution of its sensory fibers.

The hypothesis was further strengthened by the isolated occurrence of a typical case of Bell's palsy in the same ward as that in which the cases of herpes zoster occurred. ECHOLS, New Orleans. [ARCH. NEUROL. & PSYCHIAT.]

DIAGNOSIS OF CEREBRAL TUMORS. WALDEMIR SALEM, Brasil med. **55**:3 (Jan.) 1941.

Salem presented 30 cases in which cerebral tumor was diagnosed by otoneurologic examination and verified by operation or autopsy. He described the various methods of the vestibular tests, with the clinical significance of each finding. His conclusions were: (1) that vestibular tests are of great diagnostic value but that their value decreases in the presence of a marked increase in intracranial pressure; (2) that a circumscribed cerebellar lesion does not produce spontaneous nystagmus; (3) that the predominance of the slow phase of an induced nystagmus is a sign of a central lesion but is not pathognomonic of a lesion of the posterior fossa; (4) that he was able to demonstrate that the nerve fibers from the horizontal canal do not pass through the pons; (5) that a slow progressive intracranial tumor which does not produce cochleovestibular disturbances is probably not in the posterior fossa, and (6) that next to nystagmus the most important postcaloric sign is falling or oscillation. PERSKY, Philadelphia.

INFLUENCE OF THE MENSTRUAL CYCLE ON EAR, NOSE AND THROAT DISEASES. MARGARETE JENSEN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **47**:47 (Aug.) 1940.

In discussing the influence of menstruation on the normal physiology of the body, Jensen points out that it is the premenstrual and early menstrual stage which exerts an influence on the circulation. This is evidenced by increase in blood pressure, generalized dilatation of the capillary system, increase in the pulse rate and slight rise in temperature. Both the red and the white blood cells show a slight increase; also the serum sugar, globulin and lipase. There is a mild shift of the acid-base ratio to the acid side. Finally, there is vagotonia of the vegetative nervous system and an increase of the activity of the endocrine glands.

The local changes in the ears, nose and throat are evidenced by capillary dilatation, hyperemia, stasis and slight increase of blood pressure. As a result of this, there is impairment of the normal function of the mucous membranes and alteration in the calcium metabolism.

The author analyzes the data on a group of 143 patients who in the course of some manifestations referable to the ear, nose and throat passed through a menstrual cycle. Sixty-eight patients in this group presented an onset of symptoms which either suggested or produced a complication of the underlying condition. She outlines their cases in detail. The symptoms presented included vertigo nystagmus, nausea, vomiting, elevation of temperature, chills, epistaxis and cervical adenitis. Headache was a frequent occurrence. The underlying pathologic condition included simple acute otitis media, preoperative and postoperative mastoiditis, lateral sinus thrombosis, acute tonsillitis, peritonsillar abscess and acute and chronic sinusitis. In the cases of otitis the onset of symptoms, particularly of pain over the mastoid region, suggested involvement of the mastoid process. In the postoperative cases, especially in those in which submucous resection or tonsillectomy had been performed, elevation of temperature, secondary hemorrhage and cervical adenitis were recorded.

In a few cases Jensen noted that during menstruation there was rapid progression of the pathologic condition—e. g., in acute otitis media, development of fulminant necrosing mastoiditis, and in tonsillitis, development of a peritonsillar abscess. As a rule, most of these superimposed symptoms subsided rapidly after the establishment or completion of the menstrual cycle. It was often difficult to separate organic changes from subjective complaints. This was especially true when a frank pathologic state was present.

Jensen ascribes these complications to (1) lowering of the bactericidal action of the blood, (2) lability of the vegetative nervous system, with alteration in the mineral and protein metabolism, (3) loss of follicular hormone and (4) production of a so-called menstrual poison.

In conclusion, she stresses the importance of the menstrual cycle and the need for careful differentiation between real and apparent complications.

PERSKY, Philadelphia.

ROLE OF THE TONSILS AND ADENOIDS IN SCARLET FEVER. GEZA FABINYI, Ztschr. f. Hals-, Nasen- u. Ohrenh. **47**:82 (Aug.) 1940.

Fabinyi discusses the role of tonsils and adenoids in scarlet fever. He analyzed a group of 2,266 patients, of whom 209 had undergone previous tonsillectomy. He arranged his patients according to various age limits and studied factors that may influence each group.

He found that the size of the tonsils did not have any particular influence on the course of scarlet fever in patients of any age. The adenoids were grouped according to size—small, middle size and large. There was a surprising number of medium and large adenoids present in the older patients. The author stresses the importance of this finding, particularly in relation to complications. On the other hand, he found that he could not predict by the size of either the tonsils or the adenoids whether the course of scarlet fever would be mild or severe. Nor were there any indications of an influence on late complications.

Likewise, the size of the tonsils or of the adenoids has no relation to the degree of lymphadenitis which may accompany scarlet fever, although there seemed to be a tendency toward larger lymph nodes in the younger patients.

Middle ear infections were definitely influenced, more by the age element than by the size of the tonsils or adenoids. However, Fabinyi believes that there was a greater tendency toward middle ear infection in the presence of a well developed Waldeyer ring. The fact that this occurs more frequently in younger patients apparently influenced his deduction.

In comparing the tonsillectomized patients with those who had not had any previous operation, Fabinyi found that middle ear disease and its complications occur less frequently in those who have had a tonsil and adenoid operation. When these complications occur in tonsillectomized patients, particularly in those of the older age group, there is usually some residual adenoid tissue present.

In conclusion, Fabinyi states that scarlet fever has an affinity for the lymphatic structures of the pharynx and nasal pharynx, particularly Waldeyer's ring. His studies reveal the greater number of scarlet fever patients who had enlarged tonsils and adenoids. However, there is no relationship between the scarlet fever infection and the size of the tonsils or adenoids. In tonsillectomized patients the frequency of otitis media is less, its course is milder and the mastoid complications are less frequent. This is also true of other complications, such as nephritis, cardiac and pulmonary involvement, peritonsillar abscess or sinus infection. Last, when tonsillectomy and adenoidectomy were performed shortly before the onset of scarlatinal infection, the course of the latter was usually free of any complications.

PERSKY, Philadelphia.

Society Transactions

MASSACHUSETTS EYE AND EAR INFIRMARY

Cases Selected from the Regular Weekly Clinical Meetings from July 1940 to February 1941

EDITED BY ROBERT L. GOODALE, M.D., AND LYMAN G. RICHARDS, M.D., BOSTON

Hemangioma of the Temporal Bone. DR. P. E. MELTZER, DR. M. G. EVANS and DR. D. P. CORDRAY.

A white man aged 30 entered the Massachusetts Eye and Ear Infirmary on July 15, 1940, complaining of pain in the left ear and the left side of the face of two weeks' duration, facial paralysis on the left side of twelve years' duration and discharging of the left ear since the age of 3.

Two weeks before admission a diagnosis of brain abscess was made at another hospital. On his admission to the Massachusett Eye and Ear Infirmary examination showed (1) 2 diopters of choking of the optic disks; (2) complete peripheral facial paralysis on the left side; (3) a discharging left ear; (4) a small mass obscuring the drum membrane in the left canal; (5) crepitus over the temporal bony cortex and tenderness over the mastoid antrum, and (6) a spinal fluid with an initial pressure of 340 and no cells.

DISCUSSION

DR. A. S. MACMILLAN: The roentgen film showed a large cleancut cavity which might be that of a cholesteatoma. No cells remained in the bone. The blood vessels and the emissary vein were not enlarged. The posterior view of the petrous pyramid showed complete riddling of the petrous apex.

DR. D. P. CORDRAY: On July 17 Dr. Maurice G. Evans operated on the patient. The usual mastoid incision was made, and very thick sclerotic bone was encountered immediately. It was broken down in several places by what appeared to be a tumor mass. The cortex was removed, and 1 inch (2.5 cm.) of temporal dura was exposed. It was easy to see the tumor mass, which was reddish brown, with questionable pulsation. A curet passed easily down into the petrous apex. Profuse bleeding necessitated packing of the wound, and the operation was stopped. The patient was returned to the ward with a provisional diagnosis of hemangioma. High voltage roentgen therapy was instituted, and the patient received 1,200 roentgens during the first five days. Removal of the packs was attempted, the first two or three coming out rather easily. Then there was a gush of blood. The patient was taken to the operating room and the wound immediately repacked with iodoform gauze. Since immediate transfusion the patient has made excellent progress.

Two days ago Dr. G. B. Fred had the courage to remove the packing. There was not much bleeding. During the last ten days the patient has received an additional 2,000 r. The tumor seems to be much more gray. At present a very light pack is in the wound; there is no bleeding. The patient is to be discharged and to come back in one month for further roentgen treatment. The choking of the disks has remained the same.

DR. L. A. SCHALL: Without any question the lateral sinus was encompassed in this tumor. The bleeding was profuse. The operation was intended to provide drainage, and the wound was to be left open and radium given. I have 1 patient, out of 3 with hemangioma of the temporal bone, who is alive and well after six to eight years. The other 2 patients died—one two years ago, of pneumonia, the other about three years ago, of metastasis and recurrence.

Chronic Suppurative Otitis Media on the Right, with Labyrinthitis Following the Removal of an Aural Polyp. DR. L. E. WHITE and DR. JULIO QUEVEDO.

ORIGINAL REPORT

An Italian aged 43 was admitted September 29 with the following history: He was a soldier in the last World War, and after an explosion the right ear bled for a few days. He was treated and returned to the battlefield, since which time both ears have been discharging intermittently. For the past two years the hearing on the right side has been diminished considerably. The hearing on the left side also is poor, and there is a foul-smelling discharge from both ears.

About seven or eight years ago he had an operation on the left ear; he thinks this was for the removal of cerumen.

Ten days before admission the right ear started to discharge again. The patient complained of rather severe pain and headache, for which he consulted an otolaryngologist, and on September 27, two days before admission, a polyp was removed from the right ear. After that the pain was relieved somewhat but the discharge was unchanged, and the following day he began to experience dizziness, which became progressively worse and was accompanied by nausea and occasional vomiting.

On admission, examination of the right ear showed a polypoid growth completely filling the aural canal. There was a foul yellowish discharge but no

Hearing Test

	Right Ear	Left Ear
Whisper	0	6 inches
Conversation	0	12 feet
Lower level	0	64
Higher level	0	16,000
Rinne	0/60	40/55
Weber	..	→+

tenderness or swelling. The anterior-inferior part of the drum membrane of the left ear could be seen, but the rest of the drum membrane was covered by a hard substance which looked like cholesteatoma, and there seemed to be a cavity extending into the epitympanic space. The patient also had third degree rotatory nystagmus to the left side, and he past pointed 3 inches (7.5 cm.) to the right. The nose showed some septal obstruction; the throat was normal. There was no stiffness of the neck. The ocular examination gave negative results except for the nystagmus. The neurologic examination revealed nothing abnormal except nystagmus, nausea and vomiting.

The temperature has always been normal. The white blood cells numbered 16,400, with 80 per cent polymorphonuclears. On lumbar puncture the initial pressure of the spinal fluid was 300. No cells were found in the fluid. Chemical analysis of the spinal fluid showed 67 mg. of protein, 91 mg. of sugar per hundred cubic centimeters and 7.14 mg. of chlorides. Roentgenograms were taken on admission, which showed enlargement of the mastoid antrum on the left side with a definite cholesteatomatous enlargement of the antrum on the right side. Otherwise the mastoid process was sclerosed.

The patient was kept absolutely quiet in bed. The next day the initial pressure of the spinal fluid was 300, and there were 14 cells.

DISCUSSION

DR. G. L. TOBEY JR.: This patient had an acute exacerbation of a chronic discharge from the ear, with headache. When he consulted the otolaryngologist who cared for him previously, there evidently was a large polyp in the external auditory canal. A patient with chronic otitis and acute exacerbation and headache, increased discharge and obstruction to drainage in the external auditory meatus

is in a condition indicating immediate radical operation. I am much opposed to removing polyps in cases of this type, because of the frequent occurrence of labyrinthitis and meningitis following this procedure. This man should have had a radical operation immediately—not a conservative operation, such as the removal of a polyp. Whether or not immediate operation would have alleviated the labyrinthitis is unknown, but the removing of polyps often induces labyrinthitis. There is no question as to the development of progressive meningitis. My procedure now would be to perform a radical mastoidectomy on this man.

I have encountered several instances of meningitis following the removal of a polyp—2 of them my own. That is why I have always advocated hospitalization when a polyp is to be removed. A physician who removes polyps in his office is going to have to deal with a sudden labyrinthine upset. In cases in which there is an old chronic otitis with no indication of acute exacerbation and no headache or dizziness, there are proper ways of removing polyps. The only safe way is to pass a hook of horsehair in a Blake snare around the polyp and to make a clean cut through the attachment of the growth. When one engages a polyp with a small wire and gives a little pull to remove it, one can be certain that sooner or later one is going to open the labyrinth.

Unquestionably, in this case there is either a fistula in the horizontal semicircular canal or osteomyelitis of the inner wall of the middle ear. With this type of case the labyrinth should be uncapped between the round and the oval window and an opening made into the horizontal semicircular canal for through and through drainage.

DR. P. E. MELTZER: My associates and I have elected not to do anything to this patient for the time being, until the condition quiets down completely; when there is no nystagmus or other symptoms, we shall plan to do a radical mastoidectomy after five or six weeks, and discuss at that time whether to enter the labyrinth.

DR. G. L. TOBEY JR.: In five to six weeks the situation may be entirely different. If the labyrinth has died, I should drain it. We don't know what kind of surgical treatment the patient has had in his external auditory canal and whether or not there has been some injury to the stapes. There is no question in my mind that this patient should have an immediate radical mastoidectomy. From then on the otologists in charge must be guided by the findings.

FURTHER REPORT, NOVEMBER 13

DR. JULIO QUEVEDO: After admission the patient was kept under observation for a few days. He was feeling better. Another lumbar puncture showed the same pressure, but with 23 cells in the spinal fluid, of which 9 were polymorphonuclears and 14 lymphocytes. He had no nausea or vomiting. The following day the pressure of the spinal fluid dropped to a normal level of 150 and there were 6 cells, 4 of which were polymorphonuclears and 2 lymphocytes. Cultures of material from the ear showed Bacillus proteus.

On the night of October 4 the temperature went up to 101.2 F. The patient complained of pain in the ear, and the next day another lumbar puncture showed an initial pressure of 170, normal dynamics and 210 cells, of which 62 per cent were polymorphonuclears. A radical mastoidectomy was performed, and pus was found under pressure in the antrum. There was a fistula in the horizontal semicircular canal, also cholesteatomatous material. The dura of the middle fossa was exposed and the facial nerve apparently uncapped but not seen. A labyrinthectomy was performed, an electric drill being used to enter and partially destroy the three semicircular canals. An opening about 3 mm. in diameter was made in the vestibule, posterior to the semicircular canals, and the first turn of the cochlea was entered through the promontory. The wound was left wide open and sulfapyridine (2-[para-aminobenzenesulfonamido]-pyridine) administered that same night.

The next day there were 930 cells in the spinal fluid but the patient seemed slightly improved. The improvement persisted, and successive lumbar punctures showed a drop from 306 to 54 cells. The first degree nystagmus persisted.

Nine days after the operation a rash developed, which was thought to be due to the sulfapyridine which the patient received, and chemotherapy was therefore stopped. Otherwise, he has made an uneventful recovery.

DR. L. E. WHITE: We present this case to show, first of all, the danger associated with the removal of an aural polyp in the presence of an acute infection. It seems to me it would be much better in a case such as this to admit the patient to the hospital for general study rather than to attempt to remove a polyp—as I think was done in this case—in an office. We have presented this patient at several previous clinical meetings, and some members of the staff felt that it was much safer to wait, possibly for a period of three weeks or a month, before doing a radical operation, with the hope that we should thus avoid having to operate on and drain his labyrinth. Unfortunately, we were not able to do that, and I found at the time of the operation much more than the roentgenogram had led me to suspect was present.

Chronic Suppurative Otitis Media on the Right Side; Acute Otitis Media with Mastoiditis on the Left Side. DR. H. G. TOBEY and DR. R. E. MCCALL.

DR. R. E. MCCALL: An 8 year old patient entered the hospital on Dec. 27, 1940 with recurrent acute otitis media on the right side. In 1933, at the age of 1 year, he had acute otitis media on the right side, which recurred in 1935. He was first seen at the Massachusetts Eye and Ear Infirmary in 1937, with another recurrence in the right ear. At that time the left ear was normal. Later in that year, however, the left ear discharged for a while but finally became normal. The right ear continued to present a mucoid discharge.

In March 1938 he received roentgen treatment for lymphoid tissue of the nasopharynx. He was seen again on July 20, 1938 and on Jan. 24, 1940 for exacerbations of chronic otitis media on the right side. He was admitted to the hospital. On examination the left ear showed scarring of the membrana tympani. Although the condition improved with local medication, the right ear continued to discharge. The patient was discharged from the hospital for follow-up care in the outpatient department. He returned in October with mastoiditis on the left side and meningitis. At that time a simple mastoidectomy was performed on the left side. With the operation and the administration of sulfanilamide, the meningitis cleared up promptly. At operation the dura was exposed and found to be normal. A thrombus was removed from the lateral sinus. In spite of the fact that the patient continued to have an intermittent fever, the ear seemed to be improving locally.

On October 25 an alveolar abscess developed. After the extraction of several deciduous teeth his temperature became normal and the administration of sulfanilamide was discontinued. He was discharged on November 8 but was readmitted on December 27 because of what appeared to be leaking of cerebrospinal fluid from the postaural wound on the left side. This has continued until the present time but is so slight that not enough of the fluid can be procured for analysis. Both middle ears are dry. Cultures from the postaural region again show Staphylococcus aureus. The fields and the fundi are normal.

Intermittent fever continued as before, but yesterday another alveolar abscess developed.

DR. H. G. TOBEY: Not enough fluid has been obtained for one to be certain that it is cerebrospinal fluid. There was a small fistula in the upper end of the wound, but on probing, no bare bone could be felt. I think Dr. Philip Mysel did a decompression of the jugular vein, with a slight increase in the discharge of fluid. When he came back, an attempt was made to close the fistula with silver nitrate, but that has not succeeded. The next step will be to revise the wound and to see if we can find any place that is leaking and perhaps to repair it with a fascial flap. I think this patient was presented on account of the continuing elevation of temperature at a time when he had a dental infection, to which, at

the previous admission, it was felt the intermittent fever was due. At the original operation a thrombus was removed from the sinus.

DR. G. H. WRIGHT: The recurrent alveolar abscess is a focus of infection. The fact that it broke out the second time indicates that there is some involvement of the bone.

Tumor of the Ethmoid. DR. G. H. POIRIER and DR. E. F. LAWLOR.

A 38 year old woman was admitted to the hospital on Jan. 5, 1941. She was first seen January 4, in the outpatient department. She has had a discharge from her right mastoid process as long as she can remember. In July 1940 she began to have a frontal headache on the right side and increased nasal discharge, which she interpreted as symptoms of a cold. Breathing from the right nostril was at first difficult, then became impossible. The symptoms were not severe enough to cause her to seek medical care. Two weeks previous to admission she suffered a three hour epistaxis, at which time her doctor found a tumor mass in the right nostril.

On being questioned the patient told of failing vision in the right eye and of epiphora. She had lost 17 pounds (7.5 Kg.) in weight in five months.

Physical examination revealed nothing of importance. The local examination showed fulness at the inner canthus of the right eye. The vision in the right eye was 20/30; that in the left eye, 20/70. There was obvious tearing. The right nostril showed a bluish red mass filling the middle meatus, replacing the middle turbinate and extending back to the choana. The floor of the nose was filled with mucopus.

DISCUSSION

DR. A. S. MACMILLAN: Roentgen examination shows a tumor mass in the right side of the nose, with the ethmoid cell partitions and the ethmoid plate gone. The frontal sinus and the antrum are blocked. Another film shows a mass extending down into the nasopharynx. The posterior pharyngeal wall is all right. There is no destruction of the sphenoid bone. I am inclined to believe that there is destruction in the region of the cribriform plate.

DR. E. F. LAWLOR: A specimen for biopsy was taken on Jan. 6, 1941, with the region under local anesthesia, and the growth was reported as neuroblastoma. On January 14 a lateral rhinotomy was done by Dr. L. A. Schall.

DR. L. A. SCHALL: This tumor mass involved the right ethmoid labyrinth. I could not make out destruction of the cribriform plate, although the tumor mass was attached to it. With the probe it felt intact, but I did not use much pressure. The os planum of the ethmoid was entirely gone. The tumor was attached also to the orbital periosteum. It did not extend into the sphenoid sinus, which was full of pus, and, as Dr. MacMillan showed you on the roentgenogram, there was choanal extension of the tumor. We left the incision open and gave 2,000 milligram hours of radiation, although we recognized the fact that neuroblastomas are not satisfactorily radiosensitive. We expect a recurrence and believe there is still a microscopic amount of tumor attached to the cribriform plate.

Neuroblastoma of this region is extremely rare. I should call this a very rapidly growing mass.

A Case for Diagnosis: (1) Labyrinthitis?; (2) Ménière's Syndrome? (3) Subdural Abscess? DR. H. G. TOBEY and DR. R. E. MCCALL.

ORIGINAL REPORT

A boy of 21 was first seen in 1932 with a diagnosis of subacute otitis media (recurrent) on the right side, with mastoiditis. In 1924, in Chicago, he had acute mastoiditis on the right side, followed by scarlet fever. He had had a simple mastoidectomy on the right side; which was revised some time later. Then, in 1932 he entered the Massachusetts Eye and Ear Infirmary with otitis media of four years' duration, and the mastoidectomy on the right side was again revised.

He was readmitted in 1936 with a history of intermittent discharge from the right ear since the previous operation. During this time also he had intermittent headaches on the right side, with stabbing pain in the right ear and periods of vertigo and nausea. The attacks varied, occurring several weeks or months apart, lasting one or several days and becoming more pronounced. On the day of admission he stated that he tended to fall to the left side.

Examination showed no nystagmus. A lumbar puncture gave negative results. At that time a radical mastoidectomy was done on the right side, and his recovery was uneventful. He was discharged improved.

He was readmitted one month after discharge from the hospital because of a return of the headaches, dizziness and nausea. At that time he had no nystagmus and no vomiting. Granulations were present in the operative cavity. The symptoms gradually abated under observation, and he was discharged three weeks later. Until July 1937 he was followed in the outpatient department. In 1940 he again complained of dizziness, headache on the right side, a tendency to fall to the right and occasional nausea and vomiting, which were worse at night. These symptoms had been present for the previous year. He was referred for ocular and neurologic examination but failed to keep the appointments.

He was next seen Nov. 4, 1940 and was readmitted. He had a history of staggering, with a tendency to fall to the right, and some tinnitus in the right ear. There was no spontaneous nystagmus, but it was reported that the reaction to a fistula test was positive in the right ear, causing nystagmus to the left. In the Romberg test he tended to fall backward.

The hearing test showed that the labyrinth was alive. At that time the spinal fluid was entirely normal, as were the visual fields and the ocular fundi.

DISCUSSION

DR. A. S. MACMILLAN: Roentgenograms show a postoperative right mastoid with a perfectly solid petrous pyramid. There is nothing abnormal from a roentgenologic standpoint.

DR. R. E. MCCALL: This time a revision of the radical mastoidectomy on the right side was done. Both the dura and the sinus were exposed and found to be normal. The patient's symptoms improved after this operation. He was discharged December 20. He was readmitted Jan. 3, 1941, with a recurrence of the same symptoms. The operative cavity was clean except for a few granulations. There was no spontaneous nystagmus, but the fistula test still produced a positive reaction, causing second degree nystagmus to the left. A caloric test elicited a response, although this was hypoactive. Roentgenograms of the skull were made but revealed nothing of importance.

The patient improved somewhat and was discharged. On February 8 the administration of potassium chloride was instituted, and at this time the patient's symptoms were more marked when he was lying in bed with his eyes closed. On February 11 he was again readmitted, complaining of severe headache on the right side and vertigo. The patient was very restless and fell out of bed once but has no recollection of the incident. He has no hearing in the right ear. The visual fields and the ocular fundi are normal, and the spinal fluid is normal. He has no fever.

DR. H. G. TOBEY: There were very solid granulations in the posterior part of the mastoidectomy cavity. Nothing has relieved the symptoms of vertigo and headache. The question is whether we are dealing with chronic labyrinthitis with complete loss of function or with a chronic cerebellar abscess. With a chronically involved labyrinth which has finally died, there hardy seemed need for more than conservative treatment. The symptoms resemble those of Ménière's disease. Then there is the question of chronic cerebellar abscess. We are going to take this up with the neurologic department tomorrow morning.

DR. L. A. SCHALL: The episode in which the patient lost consciousness could not be explained by Ménière's disease or by labyrinthitis. The manifestations look like those of a chronic cerebellar abscess.

DR. H. G. TOBEY: The resident physician in the neurologic department seems to think that a ventriculogram will not help a great deal when the lesion is in the posterior fossa.

FURTHER REPORT

DR. H. G. TOBEY: In April 1941 the patient was seen in the neuromedical service. The impression at this time was that there was a disturbance of function in the pons and the cerebellum. However, roentgenograms of the skull failed to show evidence of tumor. On May 12, 1941 the patient still had dizziness and headache, although of less severity.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY

KARL M. HOUSER, M.D., *Chairman*

WILLIAM HEWSON, M.D., *Clerk*

Monthly Meeting, Oct. 15, 1941

GEORGE L. WHELAN, M.D., *Editor of Abstracts*

Traumatic Perforation of the Right Frontal Sinus with Pneumocephalus and Brain Abscess: Report of a Case. DR. PHILIP A. MARDEN.

M. W., a 10 year old white boy, was admitted to the Hospital of the University of Pennsylvania on Feb. 16, 1941. Two days previously, while he was coasting, an iron runner on the preceding sled had been driven into his skull at the superior inner angle of his right orbit. He extricated himself and was able to walk home unaided. A local physician closed the wound with clips. The boy was never unconscious. During the next few hours fever and vomiting developed. The wound was reopened, revealing a cerebrospinal fluid leak and traumatized brain tissue. Tetanus antitoxin (1,500 units) was given intramuscularly, and the administration of sulfanilamide by mouth was begun. He was brought to Philadelphia forty-eight hours after the accident.

Examination on admission revealed a pale frightened querulous boy, with a temperature of 102 F. and a pulse rate of 120. Cerebrospinal fluid and blood oozed from a small gaping wound at the upper inner angle of the right orbit. The globe of the right eye was intact but showed pulsating proptosis and pronounced chemosis of the bulbar conjunctiva. Flexing of the neck evoked some pain, but there was very little stiffness. There was a weak Brudzinski sign. Roentgen studies of the skull disclosed a penetrating wound at the medial superior margin of the right orbit, involving the orbital process of the frontal bone, with at least one long fragment driven into the right frontal lobe of the brain. Within the frontal lobe, in the region of the injury, a small collection of air was seen. Examination of the blood showed 21,000 white cells per cubic millimeter and the hemoglobin content 69 per cent. Cerebrospinal fluid obtained by lumbar puncture contained 621 red blood cells per cubic millimeter, no white cells and 30 mg. of protein; there was a negative serologic reaction. A smear and a culture showed the fluid to be normal, and the colloidal gold curve was normal.

The patient's past medical history included catarrhal jaundice and measles. Two rather obscure convulsive attacks one year previously were described as "worm fits." The family history revealed nothing of significance.

On the nineteenth day of hospitalization, with the patient under anesthesia induced with ether, an incision was made through the medial part of the right eyebrow. A periosteal flap was elevated. Loose fragments of bone were found

on the anterior wall, the floor and the posterior wall of the frontal sinus. These fragments, together with as much of the anterior wall as was thought necessary for the provision of adequate exposure, were removed. Hyperplastic mucous membrane and granulation tissue filling the sinus were cleaned out. When the dura was exposed, a large fragment of bone was found penetrating the frontal lobe. This was removed. A needle was directed upward into what was indicated by the roentgenograms to be the vicinity of the abscess, and about 5 cc. of pus was aspirated. A pathway to the abscess was created by a small hemostat and a 5 mm. rubber tube inserted. The incision was closed about the tube with skin clips.

During the first few days after the operation drainage was slight. Three cubic centimeters of colloidal thorium dioxide (thorotrast), a radio-opaque material, was injected through the tube under fluoroscopic control. This outlined the abscess clearly and disclosed that the end of the drainage tube projected about ½ inch (1.25 cm.) beyond the abscess cavity. The tube was then withdrawn slightly. A moderate amount of pus drained through it for two weeks, then gradually lessened. At the end of the fourth week the tube was removed, and the wound proceeded to close. When the patient was discharged from the hospital, thirty-eight days after the operation, a slight facial weakness was the only remaining neurologic sign. For six weeks all went well. Then a small fistula appeared below the medial end of the scar. On July 9 the patient was readmitted to the hospital for study.

A well healed scar lay within the right eyebrow. Below the scar and just over the inner canthus of the right eye was a soft semifluctuant nodule, 0.7 by 1.5 cm., running parallel to the margin of the lid in its long axis and covered with crusts of dried exudate. The left eye was entirely normal. The right eye presented a somewhat narrowed palpebral fissure, a round pupil with normal accommodation and convergence, no nystagmus, no exophthalmos and a normal fundus. Ocular movements revealed a slight lateral deviation on upward gaze and a lagging on clockwise motion of the right eye which caused diplopia between 2 and 3 o'clock. A slight facial weakness was present on the left side. Urinalysis and studies of the blood gave results within normal limits. There was no evidence of increased intracranial pressure or any signs of osteomyelitis of the bones of the calvaria.

The administration of sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) by mouth was instituted, and on July 23 the fistula was explored. In spite of an extensive search no sequestrum was found in the frontal lobe. The external portion of the fistulous tract was excised and a large rubber drainage tube inserted. This tube was left in place for one week and then removed. The wound healed promptly. The postoperative course was uneventful, and the patient was discharged from the hospital eighteen days after the operation.

For two weeks following discharge from the hospital the wound remained closed. Then, as before, a small red nodule appeared below the scar, soon breaking down with the formation of a fistula. Colorless fluid has drained intermittently until the present time. The patient has continued to feel very well. He has had no headache, stiff neck or impairment of vision.

On October 6 the patient was admitted to the hospital for the third time. His general appearance was excellent; his temperature and pulse were normal. A slight facial weakness on the left side was the only remaining neurologic sign. Below the medial aspect of the right supraorbital cicatrix a thick crust was found covering a small mass of granulation tissue. On removal of this crust a few drops of serosanguinous fluid issued from the center of the granulation tissue. Studies of the blood and urine revealed normal conditions. Cerebrospinal fluid obtained by lumbar puncture was likewise normal. In view of the most recent roentgen studies, the roentgenologists believe that the shadow which was thought to be that of a bony sequestrum in the frontal lobe of the brain is caused by the retention of radio-opaque material employed previously to outline the sinus and the abscess cavity. The problem of the successful closure of the fistula from the frontal sinus still remains, and my associates and I are now (October 22) planning steps to achieve this end.

Extensive Carcinoma of the Maxillary Sinus. DR. C. CALVIN FOX.

A white man aged 50 entered the hospital with the chief complaints of (1) pain and swelling in the right side of the face for a period of seven months and (2) protrusion of the right eye for five months. During the early stage of these symptoms the right antrum had been irrigated without benefit. There was a gradual increase of swelling, pain and purulent nasal secretion. Vision and ocular motion were impaired. A loss of 40 pounds (18 Kg.) of weight was accompanied by weakness. There was a firm induration and swelling of the right cheek. It involved the lower border of the orbit, causing the eye to be elevated, proptosed and almost immobile. The optic disk was blurred and the fundus edematous. The vision in the right eye was 20/200. Roentgen studies showed an extensive malignant growth involving the right antrum, orbit and ethmoid labyrinth.

A radical maxillary sinus and transantral ethmoid sinus operation was done through a supra-alveolar incision. The lateral, superior and medial antral walls were necrotic, and the pterygomaxillary space was invaded. All available tumor was removed, and 3,450 millicurie hours of radium were employed. Nine days later a course of 4,800 roentgens (r) of radiation therapy was started. The buccoantral wound was kept open.

There were pronounced postoperative swelling and edema in the face and eye. This disappeared almost entirely in four weeks. The patient left the hospital eight and one-half weeks after operation. In six months the face had a sunken appearance. The eye was somewhat divergent, the eyeball fixed. The lens showed opacity. Some papilledema was present. In the right eye there was vision only for finger movements. The antral wound was kept open, and some crusting was cared for by irrigating with a saline solution. Roentgen examination seven and one-half months after operation showed definite improvement of the antrum and no evidence of a malignant condition.

In September 1941, three years after operation, a thorough study showed the patient to be in good health, having gained 40 pounds (18 Kg.). Roentgen study revealed no malignant condition On two occasions during the past three years biopsy did not show carcinoma. There is practically no vision in the right eye, where an incipient cataract is about the only ocular change observed.

Massive Osteomyelitis as a Complication of Pansinusitis. DR. SAMUEL R. SKILLERN JR.

This case provides a perfect demonstration of the ravages of rapidly spreading osteomyelitis complicating a long-standing sinus infection. The erroneous advice was given that as the sinus infection was of allergic origin, all surgical treatment of the sinuses should be ruled out. The failure to differentiate bacterial allergy from that associated with more common factors, such as house dust, plant pollen, food proteins and inorganic substance, was in all probability the inception of the incorrect advice and its tragic sequela.

Allergy of pathogenic origin within the nasal sinuses which does not respond to systemic or local treatment requires surgical intervention for the removal of the foci of infection. In allergy of nonpathogenic organismal causation surgical intervention is generally contraindicated.

Dr. H. Leon Jameson has expressed the opinion that in many cases chronic septic bronchiectasis is due to an overlying sinus disease.

The patient gave a history of allergy since childhood; sinus infection since early manhood, with hemoptysis, and a diagnosis of bronchiectasis in 1935.

His pansinusitis was complicated by such massive organismal invasion of the bones of the forehead, temple, cheeks, orbital cavity and nasal areas that a single stage operation was out of the question.

To complicate his prostration, generalized purulent bronchiectasis prevented placing him in a prone posture at any time. A preliminary evacuation of the pus (1 pint [473 cc.]) by bronchoscopic procedure allowed fuller respirations, but it was necessary to repeat this procedure twice during the first operation.

The first operation consisted of enlargement of the fistula in the floor of the left frontal sinus, removal of the sequestrated nasal and right malar bones and injection of a sulfathiazole paste into the underlying sinus cavities. At a later date the entire frontal bone was removed in toto. The dura appeared healthy.

A third operation was undertaken in which a large section of each temporal bone was removed. The right orbit was then enucleated, exposing the floor of the frontal sinus and the ethmoid, sphenoid, lacrimal, malar and maxillary plates forming the orbital cavity. These plates were involved in the osteomyelitic process, so that their removal was required. Some sections came away as sequestrums; others had to be removed with the Rongeur forceps.

The entire malar bone and approximately that portion of the maxillary bone forming the wall of the right maxillary sinus were removed.

Even with such horrible, sickening surgery, Dr. Coates and I felt that a similar procedure would be required on the left side of his face and calvarium. Fortunately "repossession by his Maker" saved us the necessity of such cruel surgical intervention.

Autopsy.—The postmortem observations were: massive purulent pansinusitis; osteomyelitis of facial, nasal and anterior calvarial structure; diffuse multiple and multiloculated bronchitic abscesses, with the usual degenerative processes of the adjacent vital organs.

Book Reviews

Diseases of the Ear, Nose and Throat. Principles and Practice of Otorhinolaryngology. By Francis L. Lederer, M.D., F.A.C.S., Professor and Head of the Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine; Chief of the Otolaryngological Service, Research and Educational Hospital, Chicago. Third revised edition. Illustrated with 730 halftone and line engravings on 463 figures, and 18 full page plates. Price, $10. Philadelphia: F. A. Davis Company, 1942.

The third edition of Dr. Lederer's book has just come off the press. It is a volume which retains in every manner the excellence of the previous editions. The arrangement of the material, the fine literary style, the two columns on each page, the drawings, schemata, format, binding and kindred important points have been considered fully and discussed in these columns before.

The present volume brings down to date three important chapters—namely, those on otogenic complications, labyrinthitis and blood disturbances—and some small additions have been made to other chapters.

Otogenic complications are fully covered in twelve pages, the previous edition having contained but three. There are now additional charts, with descriptive explanatory notes; tables; temperature charts; a figure describing circumscribed and persistent headache which could be due to an infection or an abscess in the frontal or the temporal lobe of the brain, an extradural abscess or an abscess of the posterior fossa; an evaluation of early signs of acute and chronic otitis in children and in adults; studies of temperature in some other otogenous complications, and a fine illustration of the mechanism of otitic complications. Therapy is fully discussed.

Labyrinthitis is covered in ten pages, whereas the previous edition contained six on this subject. There is a fuller explanation of the various stages of labyrinthitis, with an additional discussion of the postoperative stage and of the prognosis and treatment in this disease.

The previous edition allotted six pages for the discussion of blood disturbances; the present one contains eleven pages. The subject of anemia is taken up in greater detail, with fuller consideration given to pernicious anemia, aplastic anemia and the acute hemolytic anemia occurring during the adminstration of sulfanilamide. There is a general discussion of polycythemia. Purpura is more fully dealt with, and the therapy indicated for conditions that may arise during the medical or surgical treatment of any otologic condition is given more attention in this edition than in the previous one. This is in line with the constantly increasing knowledge coming from numerous sources.

This textbook is recommended to all otolaryngologists and to residents, medical students and specialists whose fields are closely related to otolaryngology.

Dr. Lederer is to be congratulated for producing such an excellent book. It is valuable for reference and for teaching purposes.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gordon F. Harkness, 215 Main St., Davenport, Iowa.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Atlantic City. Time: June 8-12, 1942.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Ralph I. Lloyd, 14–8th Ave., Brooklyn.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: San Francisco. Time: Oct. 11-15, 1942.

American Broncho-Esophagological Association

President: Dr. W. Likely Simpson, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.
Place: Hotel Dennis, Atlantic City, N. J. Time: June 8-9, 1942.

American Laryngological Association

President: Dr. T. E. Carmody, 277-16th St., Denver, Colo.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Place: Atlantic City, N. J. Time: May 25-27, 1942.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James A. Babbit, 1912 Spruce St., Philadelphia.
President-Elect: Dr. James G. Dwyer, 375 Park Ave., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Atlantic City, N. J. Time: June 1-3, 1942.

SECTIONS:

Eastern.—Chairman: Dr. Henry P. Johnson, 32 Deering St., Portland, Maine.

Southern.—Chairman: Dr. William L. McDougall, 915 Doctors' Bldg., Atlanta, Ga.

Middle.—Chairman: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis, Mo.

Western—Chairman: Dr. Emil F. Tholen, 1136 W. 6th St., Los Angeles.

American Otological Society

President: Dr. E. M. Seydell, 107 W. Douglas Ave., Wichita, Kan.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.
Place: Atlantic City, N. J. Time: May 28-29, 1942.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.